THE EFFECTS OF LUMBAR EXTENSOR FATIGUE EQUIVALENT TO SOCCER ON SPRINT KINEMATICS AND HAMSTRING TORQUE, AND THE IMPLICATIONS FOR HAMSTRING STRAIN INJURIES

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ABSTRACT

Core training is prevalent in injury prevention programmes and particularly in soccer. The anterior core muscles (i.e. muscles that flex the trunk) and posterior core muscles, particularly the lumbar extensors, possess mechanisms that increase anterior rotation of the pelvis whilst sprinting if they were weak, thus increasing hamstring injury risk. The literature indicates the lumbar extensors are more likely to be weak from soccer match play based on the demands of sprinting, which might be partly because the lumbar extensors are unable to be strengthened through typical resistance exercises. This thesis found lumbar extensor strength in soccer players is no different to resistance trained individuals and powerlifters. Further, the lumbar extensors were observed to experience a greater magnitude of fatigue compared to the trunk flexors after simulating 90 minutes of competitive soccer match play (median and interquartile range of −13% [5.5%] and −4.5% [29%] respectively), which was likely due to a local mechanism based on the observed reduction in hand grip strength (−6% [12.8%]). Therefore, it was investigated whether fatigue in the lumbar extensors equivalent to that induced from soccer match play could anteriorly tilt the pelvis whilst sprinting. The thesis established a protocol to replicate the lumbar extensor fatigue whilst avoiding fatigue in other muscles. Subsequently, this protocol was used to establish the effect of the lumbar extensor fatigue on the anterior pelvic tilt whilst running. In addition, it was investigated whether lumbar extensor fatigue can reduce hamstring torque by limiting the lumbar extensors capacity to oppose the pelvic rotation created by maximal hamstring actions. The principal findings showed the lumbar extensor fatigue significantly increased anterior pelvic tilt during the early and terminal swing phase of running, and reduced maximum hamstring torque up to -26 N·m ± 27 N·m. These findings are the first to show that weakness in a core muscle is capable of increasing anterior pelvic tilt whilst running, likely increasing the risk of hamstring strain injury. Likewise, it is the first to show evidence of proximal muscle fatigue affecting hamstring torque production. Nonetheless, the magnitude of the increase in anterior pelvic tilt (no greater than ~1.3 ± 2.0°) raises some concerns for the wider adoption of core muscle training for hamstring injury prevention.

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GLOSSARY

Abbreviation	Full term
BF	Biceps femoris
CI	Confidence interval
COM	Centre of mass
CSA	Cross sectional area
EMG	Electromyography
EO	External oblique
ES	Erector spinae
FL	Fascicle length
GRF	Ground reaction force
HSI	Hamstring strain injury
ILEX	Isolated lumbar extension
IO	Internal oblique
LIST	Loughborough intermittent shuttle test
MF	Multifidus
MRI	Magnetic resonance imaging
MTJ	Myotendinous junction
PS	Psoas
QL	Quadratus lumborum
RA	Rectus abdominis
ROM	Range of motion
SAFT90	Soccer-specific Aerobic Field Test
SD	Standard deviation
SI	Strength index
SM	Semimembranosus
ST	Semitendinosus

SECTION 1:

INTRODUCTION TO THE THESIS

1.1. OVERVIEW OF THE PROBLEM

Practitioners, coaches, and athletes are tasked with improving performance and minimising injury risk through training programmes. Inevitably some exercises must be chosen over others given the finite time available. The idealistic programme would have rigorous evidence supporting each decision, but this is not the case. In the search of the optimal programme, exercises are often chosen from clinical reasoning in the hope of maximising the benefits (Winters 2018). There is no known harm from including novel exercises (assuming appropriate execution), but their precedence over evidence-based exercises can be at best unproductive and at worst harmful (Buchheit *et al.* 2018). Of course, some postulated exercises may be beneficial, but this cannot be known, and the approach is akin to gambling with athletes' mental and physical health. For example, Injuries lead to physical maladaptations such as increased body fat and risk of future injury (Carling and Orhant 2010; Verrall *et al.* 2006) but the mental consequences are profound and can lead to early retirement (Smith *et al.* 2017). Seventy-one percent of hamstring injured athletes feared re-injury (Skaara *et al.* 2013), and among 307 retired soccer[1] players, 42.3% attributed retirement to injury, which is worsened by the fact depression is commonplace with early retirees (odds ratio [OR] = 3.44; Sanders and Stevinson 2017). Injured soccer players consider themselves 'worthless' and 'very anxious', and are unable to sleep (Wood, Harrison and Kucharska 2017; Yoon and Yoon 2014). In sports such as soccer, team performance suffers from injuries adding to the burden of athletes (Hoffman *et al.* 2019; Eirale *et al.* 2013; Hägglund *et al.* 2013), let alone the financial strain they place on clubs (Ekstrand 2013). Unfortunately, programmes and interventions that lack empirical support, and thus gamble between success and injury, are prevalent in approaches to hamstring injury prevention.

Hamstring injuries are an injury prevalent in a multitude of sports. For example, a study in 1989 in the National Football League reported 1,716 hamstring strain injuries (HSIs) over the following decade, with up to 210 occurring per season (Elliott *et al.* 2011). Soccer seems particularly susceptible to HSIs. A study in Austrian soccer identified HSIs as the most common non-contact injury

[1] 'Soccer' is preferred to 'football' or 'association football' due to its universal understanding and historical origins (Szymanski 2014).

(47% of all non-contact injuries) in a mixture of amateur and professional teams (Fischer *et al.* 2019). In a professional Spanish soccer team, HSIs were the most common injury over a single season (0.75/1000 hrs; Raya-González *et al.* 2018), although a study of greater duration in a professional Spanish team reported even greater incidence at 1.52/1000 hrs (confidence interval [CI] = 1.18–1.96/1000 hrs; Larruskain *et al.* 2018a). This resulted in 864 days lost over the five year study period and equates to ~173 days or 25 weeks lost per year (Larruskain *et al.* 2018a). Assuming 0.73 games are played per week (38 games over 52 weeks) then for this team, HSIs caused players to miss ~18 games a year.

Between 2001–2014, hamstring injuries have increased 4% annually in men's professional soccer (~32 injuries per season; Ekstrand, Waldén and Hägglund 2016) despite the known preventative benefits eccentric exercise (Schache 2012; Petersen *et al.* 2011; Arnason *et al.* 2007; Askling, Karlsson and Thorstensson 2003). This increase in HSI rates is continuing with the latest evidence showing an incidence of 3 HSIs per 1000 hrs in Australian soccer (Whalan *et al.* 2019), which is twice as many as Larruskain *et al.* (2018a) and four times more than Raya-González *et al.* (2018). There are claims that this is due to increased exposure to high-speed running rather than poor injury prevention programmes (Buchheit *et al.* 2018), but regardless, better prevention is clearly needed to ensure the health of soccer players and all athletes at risk of HSIs, particularly if the physical demands of sports have increased.

Soccer is a unique sport in that participation continues from childhood through to adulthood, and frequently appears within the top five participated physical activities across the world (Hulteen *et al.* 2016; Dvorak *et al.* 2004). Thus, preventing HSIs in soccer is key considering the health and financial benefits associated with its participation (Milanović *et al.* 2019; Oja *et al.* 2015; Ekstrand 2013). Part of soccer's appeal lies in the practically immeasurable combinations of physical, technical, psychological, and geographical aspects that creates paths within a complex system which proceedings may never follow again. Every soccer match is different. Some have more sprints, some have more tackles, and some will have more jumps. Accordingly, the demands of soccer vary between games (Carling *et al.* 2016; Bradley *et al.* 2011; Gregson *et al.* 2010; Di Salvo *et al.* 2009),

Yet, the basic physical components of soccer are clear to any observer: speed, 'power'[2], balance, and dexterity (Mumford 2019). Athletes consistently cover long distances and perform repeated sprints (Castagna *et al.* 2017; Dalen *et al.* 2016; Bradley *et al.* 2009) and often have insufficient rest between matches (Ekstrand, Spreco and Davison 2018) regardless of sex (Mara *et al.* 2017a; Mara *et al.* 2017b) and level of performance (Vigh-Larsen, Dalgas and Andersen 2018; Bradley *et al.* 2010). It is these commonalities that allow practitioners to extrapolate and take their programmes further than the evidence allows. Indeed, Buckthorpe *et al.* (2019) acknowledged that their approach to HSI prevention in professional soccer is built on limited evidence and primarily anecdotes from personal observations.

Buckthorpe *et al.* (2019) recommended HSI prevention programmes focus upon four areas: hamstring strengthening, monitoring workloads, lumbo-pelvic stability, and training movement patterns, but the evidence for their inclusion is insufficient and they might be unproductive, or even harmful. For example, whilst a large acute to chronic workload might cause hamstring injury (Duhig *et al.* 2016), it has been suggested that HSI incidence during training has increased because of attempts to increase the chronic workload in athletes (Eirale 2018). Likewise, improving lumbo-pelvic stability may well reduce injury risks as proposed by Buckthorpe *et al.* (2019), but the specific movements and muscles involved are limited to theoretical conjecture that is yet to be tested. In addition, the evidence supporting the use of movement pattern training seems to only reflect the specificity principle of strength training (Cacchio *et al.* 2007). In support of movement pattern training, Buckthorpe *et al.* (2019) refer to a simulation study that found increasing the force of muscles decreased jump height until the simulation was re-optimised (Bobbert and van Soest 1994) but this assumes the body is not capable of self-optimising and needs training for optimal mechanics. In reality, it is unlikely that the same precise mechanics persist when strength increases over time compared to an instant increase in force from 5 to 20% as per the simulation. Indeed, Moore, Jones and Dixon (2012) found that beginner runners self-optimised their kinematics after prescribing a programme of continuous running. Maas and Vanwanseele (2019) found no difference in kinematics

[2] The term power is used because of its ubiquity but is referring to impulse as per (Winter *et al.* 2016).

after a longer training duration and larger sample size but their comparisons were limited to peak angles across the gait cycle, whereas Moore, Jones and Dixon (2012) found joint angles did not change at their peaks but instead at phases of the gait cycle (i.e. toe-off). Clearly many approaches to HSI prevention need further research to ensure their efficacy. By doing so, practitioners could make more informed decisions over the utility of an approach, be more productive, and ultimately more effective. Though core strengthening in particular is thought be of great importance (Willson *et al.* 2005) and has even been referred to as 'the centrepiece' of training (Bliss and Teeple 2005). Indeed, core strengthening is among the top three injury prevention measures used by elite UEFA club teams (McCall, Dupont and Ekstrand 2016). Globally, core strengthening was reportedly used by 100% of 44 professional clubs compared to 79.5% of clubs that used eccentric training (an approach supported with evidence; McCall *et al.* 2014). More recently, 100% of clubs in the first division of Brazilian soccer implemented core training to prevent injury but eccentric training was rated less than half the importance of core strengthening (Meurer, Silva and Baroni 2017). When asked to include a 10-week Nordic curl programme, only 13% of club-seasons (total seasons across all clubs) did so, and 57% of club-seasons did not use the intervention at all despite recognising its effectiveness. In contrast, core strengthening was implemented in 100% of seasons (Bahr, Thorborg and Ekstrand 2015).

The widespread adoption of core training would suggest that it is viewed as a potential measure to prevent HSIs but evidence to support this claim is absent (Shield and Bourne 2018). It is widely believed that core strengthening can prevent lordosis and anterior pelvic rotation, which is thought to lengthen the hamstrings and increase injury risk (Rivera 2016; Mendiguchia and Brughelli 2010; Panayi 2009; Devlin 2000). The following section will review the origins of this conjecture.

1.2. AN APPRAISAL OF THE ORIGINS OF THE RELATIONSHIP BETWEEN THE CORE AND THE HAMSTRINGS

On January 1st, 1954, Overton and England (1954) published their observations of a hamstring injury case, concluding that the cause of injury was the forcible flexion of the trunk whilst the knee was extended. Within nine months Tucker and Alexander (1954) had attributed this mechanism to

inefficient trunk stabilising muscles. Their conjecture was an implicit suggestion that HSIs occur due to excess hip flexion and not knee extension. Clearly the idea that the core[3] induces hamstring injuries via hip flexion is as old as our understanding of the injury itself.

The first empirical study to refer to HSI risk factors can be traced to an investigation by Burkett (1970), who referred to Klafs and Arnheim's 1963 conjecture that implicates postural control, technique, flexibility, and strength imbalances as risks for hamstring injury (Arnheim and Prentice 1999). Yet, Burkett (1970) did not measure posture related variables in their investigation and instead measured strength and flexibility across the knee flexors and extensors. It is as if Burkett (1970) had assumed the role of postural control and technique in the mechanism of HSIs. An assumption that is widespread, as evidenced by the global adoption of core training. An apparent difference in strength between injured and healthy athletes identified by Burkett (1970) led to further research seeking to support the rationale of strength as a risk for injury, which claimed the ratio of knee flexion to extension strength is a risk factor after two athletes sustained injuries out of the five who had less than the mean hamstring strength (Christensen and Wiseman 1972). Later, Liemohn (1978) measured strength ratios between limbs but was unable to discern risk factors for injury as no inferential statistics were used. It nonetheless added to the attention and momentum strength received as a risk factor during this time, which seems to have directed the research in this field leaving postural control as an assumed risk.

In 1982, Muckle (1982) stated their belief that the lumbar spine is associated with HSIs, and one year later the first evidence of a relationship was provided by Watson (1983), who identified 43% more groin strain injuries (six injuries) in those with more lordotic postures compared to those without. Whilst these injuries were not necessarily HSIs, Watson (1983) proposed the lordotic posture resulted from anterior pelvic rotation and shortened hip flexors and lengthened hamstrings, echoing

[3] There is no consensus as to what muscles form the core (Shield and Bourne 2018). Reference to the core muscles will include any muscle that acts on the lumbar spine, pelvis and hip until a more specific definition is provided in the literature.

the thoughts of Tucker and Alexander (1954). Furthermore, Cibulka *et al.* (1986) proposed hamstring injured athletes have greater anterior rotation of the pelvis based on sacroiliac joint manipulation improving hamstring peak torque, although the absence of a control group and retrospective design limits the strength of these inferences and the evidence would still only support an anatomical association between posture and the hamstrings. Adding further doubts, another study found 76% of Grade one HSIs displayed neural tension signs (Kornberg and Lew 1989), and therefore the association between the lumbar and hamstrings might not reflect a strain injury risk. Whilst it should be considered that the frequency of a positive neural tension was not compared to healthy players (Kornberg and Lew 1989), the absence of prospective studies between posture and injury could not eliminate this possibility. In the meantime, hamstring strengthening had already been identified as a successful intervention to reduce HSI rates (Heiser *et al.* 1984) yet the evidence supporting posture and hamstring injury continued to lack substance. On reflection, it is easy to see how strength has garnered much attention as a risk factor in the present day.

After 39 years of research, the first statistical association between posture and hamstring injury risk was reported. Hennessey and Watson (1993) identified excessive lordosis in hamstring injured athletes compared to controls, which was later confirmed in relation to all lower limb injuries (Watson 1995). The excessive lordosis was thought to originate from anterior pelvic rotation, which lengthens the hamstrings and is suspected to increase injury risk. These findings align with two cases of hamstring injury where participants presented with hyper-lordosis and anterior pelvic tilt, among other postural deficits that were accompanied by increased core muscle tone according to the authors (Hoskins and Pollard 2005). Similarly, these injuries were successfully treated through a holistic treatment of the core muscles (stretching and strengthening) and spinal manipulation; Hoskins and Pollard 2005). Further work showed the severity of injuries (days lost) was associated with an index of posture related variables ($r = -0.41$; Watson 2001) such as kyphosis, head position, scoliosis, knee hyper extension, and foot arch to name a few, but it's unclear which measures of posture could be responsible for injury and which simply co-occur with one another.

Hoskins and Pollard (2005) offered an explanation for why individuals have more anterior rotation of the pelvis by referring to Janda, Frank and Liebenson's (1996) lower cross syndrome, which proposes an imbalance between erector spinae and abdominal tonicity, and the iliopsoas and gluteus maximus tonicity, resulting in anterior rotation of the pelvis. However, Janda, Frank and Liebenson (1996) refer to muscle tone originating from either the muscles visco-elastic properties or their active state. Therefore, 'tight' erector spinae muscles could be balanced through activation of the abdominals, but there is no mention of this solution. Psoas flexibility (hip extension range of motion [ROM]) was found to be not correlated to pelvic tilt or lumbar lordosis (−0.09 and 0.27 respectively) and neither was abdominal strength (−0.04 and 0.30 respectively; all $p > 0.05$; Heino, Godges and Carter 1990). Walker *et al*. (1987) reported similar findings between abdominal strength and pelvic tilt (r = 0.18) and lordosis (r = 0.06). Furthermore, lower cross syndrome implies a chronic change to posture, gradually lengthening the hamstrings over time rather than a sudden strain. Franchi, Reeves and Narici (2017) demonstrated the hamstrings adapt to chronic forceful lengthening by increasing fascicle lengths (FL) and eccentric strength, so a chronic postural change seems unlikely to increase injury risk. Such criticisms can be applied to all research using static posture as a measure of injury risk. Despite this, a sudden anterior rotation of the pelvis would indeed place the hamstrings under further strain that it would not be adapted for.

In 2004 the case for postural control causing injury was rejuvenated by Sherry and Best (2004) who performed a randomised controlled trial of agility training, core muscle strengthening, and proprioceptive exercise that limited re-injury incidence to 7.7% compared to a stretching and strengthening group with a re-injury incidence of 70% over a one year follow up (6 more injuries). This was despite the stretching and strengthening group spending longer in rehabilitation (additional 14 days) and with a smaller proportion of severe injuries at baseline. Unfortunately, the precise cause of the reduced injury rate cannot be discerned between core strengthening, agility, and proprioceptive training. Though it is the first study to prospectively associate core strengthening and hamstring injury risk. By now the research base was firmly established in hamstring strength. This was likely facilitated by the wide acceptance of deviating running kinematics (i.e. postural control)

as a cause of injury despite a dearth of evidence (Powell *et al.* 1986), and the ease of measuring hamstring strength in comparison to the core, which itself is hard to define. For example, a double-blind retrospective study concluded trunk extension and flexion 'dysfunction' is not a cause of HSIs but measured 'dysfunction' arbitrarily with little theoretical support for the measurements (Wallden and Walters 2005). More objective measures such as magnetic resonance imaging (MRI) have identified an association between small multifidus cross sectional area (CSA) and injury severity (> 4 days) in the lower limbs (2 of 12 were HSIs) that was not present for the quadratus lumborum, psoas, and transversus abdominis (Hides *et al*. 2011). This appears to be the first study to identify a core muscle that might be responsible for injury with empirical support.

The theoretical foundation which postural control is built on is that tonicity in certain muscles can cause an imbalance leading to anterior rotation of the pelvis, in turn lengthening the hamstrings. This seems unlikely to increase injury risk as the hamstrings would adapt to the chronic lengthening (Franchi, Reeves and Narici 2017). Nonetheless, it's possible insufficient core muscle strength could result in an acute increase in anterior rotation of the pelvis, lengthening the hamstrings that the hamstrings have not adapted to. Unfortunately, research regarding core muscles and postural kinematics as risk factors for hamstring injuries is lacking (Shield and Bourne 2018; Mendiguchia and Brughelli 2010). Yet at no point does there appear to be a finding or comment so profound that justifies the absence of research towards the core. Considering the global acceptance of core strengthening as measure of preventing HSIs, the impact of research in this regard would have wide-spread implications for training.

1.3. THESIS MILEU AND DIRECTION

Hamstring injury prevention programmes that implement core training are void of sufficient supporting evidence and their inclusion could compromise the delivery of proven hamstring injury prevention exercises, or wider injury prevention exercises and performance gains. This gap in knowledge is one of the barriers that must be traversed to achieve an optimal hamstring injury preventing programme. To assess whether core muscle training could reduce injury risk, it must first

be identified the whether weakness in a core muscle has a role in the mechanism of hamstring strain injury. Failing to identify this before investigating interventions can lead to invalid conclusions from unknown confounding variables and potentially an inefficient use of resources. Therefore, a literature review will be carried out to conceptualise the mechanism of HSIs and subsequently to postulate whether weakness in core muscles could increase injury risk according to the model of injury. The theory surrounding causal inference and the methodical implications will be considered before progressing to ensure valid conclusions are achieved.

The conception of the injury mechanism will be composed of two parts. Initially, the cause of strain injuries at the myofibril level will be considered. Understanding the fundamental causes of HSIs will support the reasoning used in the second part, where the literature surrounding hamstring strains specifically will be reviewed. Together they will inform the conception of a hamstring strain injury mechanism. This concept will then serve as a framework to identify which core muscles have the potential to alter the mechanism and therefore increase the risk of injury. It will be shown in the literature review that the erector spinae, or more specifically the lumbar extensors, appear the most likely to be weak and increase injury risk by inducing a forward lean and anterior pelvic tilt. Though trunk flexor weakness also possesses a mechanism to increase HSIs as their anatomical connections to the pelvis mean their actions produce posterior rotation of the pelvis. These two muscles and their potential mechanisms form the basis for the empirical studies to proceed. The thesis structure, outlined in figure 1, is intended to show the logical progression between hypothesis generation and hypothesis testing.

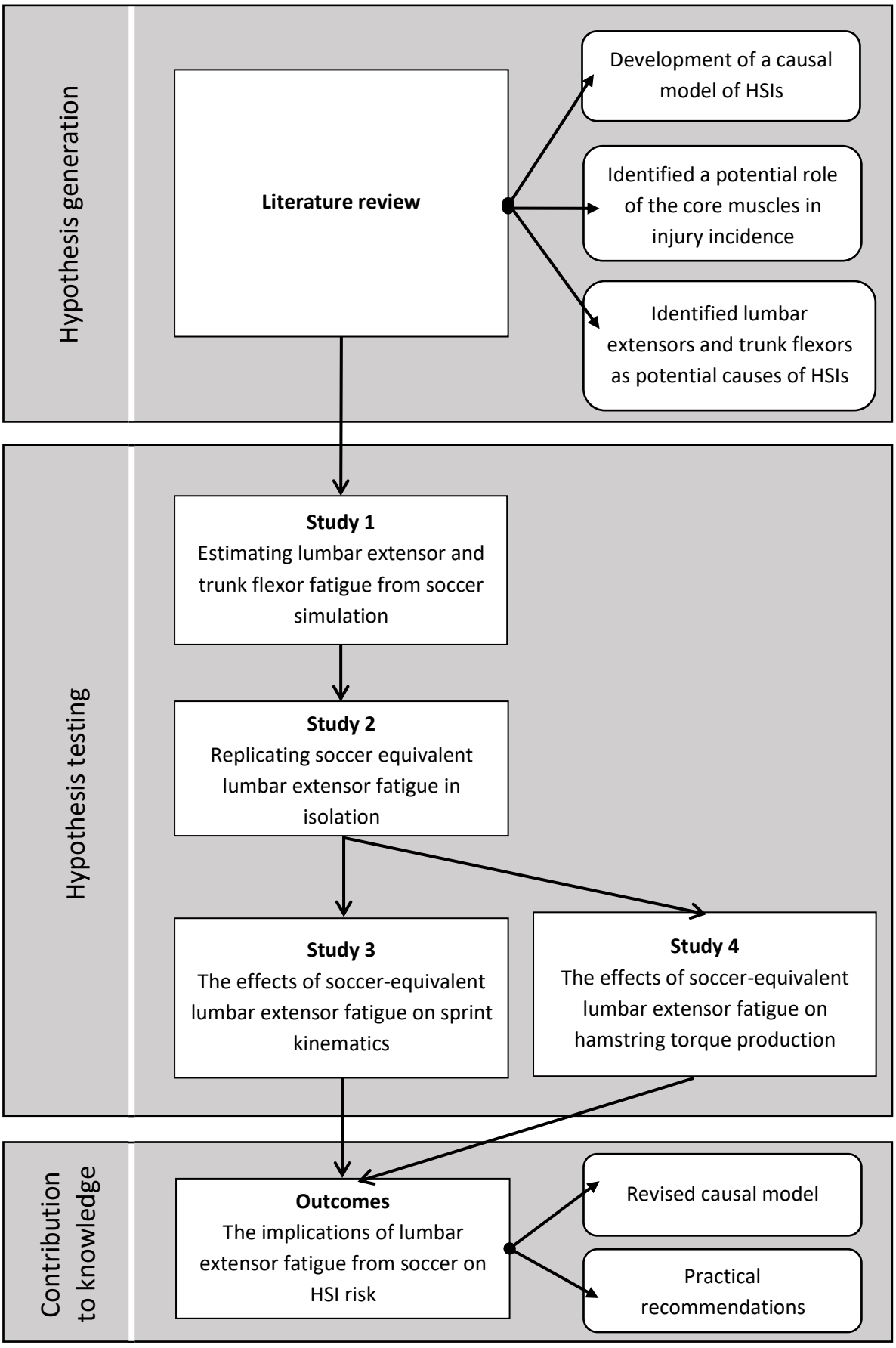

Figure 1: An outline of the thesis milieu and direction

1.4. CAUSALITY AND METHODICAL CONSIDERATIONS

Often the purpose of causal inference is to identify variables that can be intervened on to reduce the probability of an undesirable event, such as injury. The simplest way to prevent an injury from occurring is to hinder the path between cause and effect. Causal paths are a common phrase in causality research, but it may be better to view these as slides. If one enters a slide, they inevitably reach the bottom and likewise if a cause is present then the effect will inevitably occur. Causal paths imply one could stop progressing to the effect, but travelling back in time is not yet possible. What can be altered is how hard you land (the magnitude of effect).

It could seem odd to say that effects are inevitable. For example, one could sprain their ankle at the end of the slide, but this is obviously not certain, and therefore counter to logic the slide is not considered a cause of the ankle sprain. This unreasonable conclusion results from measuring effects as categorical outcomes that are not sensitive to changes in the underlying variables. Effects are always inevitable but may only be seen using methods that are sufficiently sensitive to detect them. Therefore, causality research should view effects as the underpinning continuous processes rather than the observable effect of interest that may or may not occur. Specifically, it is the change in the continuous process that are of interest (magnitude of effect).

As one progresses down the causal slide and towards the effect, they may be given an extra push to ensure a harder landing (magnitude of effect). These pushing forces are better known as mediating variables and are necessary and unique to each slide (though not necessarily exclusive). Therefore, if one wishes to reduce their landing (magnitude of effect) they should seek to weaken the pushing forces. The energy providers for these pushing forces are known as moderating variables. The more energy they offer, the more force can be used to push one down the causal slide. Therefore, reducing the available energy to a pushing mediator would lessen the magnitude of effect. Each mediating variable has its own pool of energy (set of moderators) and therefore each may need to be specifically targeted. For example, muscle damage (the effect) may be worsened with increased

muscle lengthening (the mediator), but the extent of muscle lengthening might depend on various factors, such as fascicle length and tendon stiffness (the moderators).

It is clear then, that although causes are necessary for an effect, the magnitude depends on a combination of moderating variables. It follows that the most effective interventions can only be known by specifying the causal path and their moderating variables. An alternate method to prevent effects is to remove the initial cause so that one cannot enter the causal slide. This is not usually possible, as causes are just mediating variables (a push) where the preceding cause has not been specified, usually because it is essential to the context. For example, causes such as ground reaction forces (GRFs) are necessary for sprint performance. Thus, to reduce HSIs through this event would also require one to sprint more slowly. Clearly not feasible for most athletes.

1.4.1 Specifying the causal path

In order to lessen the magnitude of an undesirable outcome, interventions must target moderating variables. Therefore, identifying these is critical. Moderating variables are often identified using observational data by estimating the risk of an event using the prevalence of an outcome between samples with and without the proposed moderator. For example, the rate of HSIs in those with a previous injury and those without (Green *et al.* 2020). Therefore, whether a moderator is deemed a cause depends on whether the categorical event occurs. Therefore, this approach only considers moderators as causes if they alone suffice to achieve the critical mass of causal power that corresponds to a morphological change. Thus, moderators that increase the probability of an event, without causing the event, are not considered causes in this approach. Statistical methods such as logistic regression provide evidence of contributory causes by providing the probability of an outcome being observed for a given moderator value. Whilst useful, such approaches cannot be relied on as it depends on the categorical effect occurring. For example, a change in a moderator may well increase the probability of an event occurring by adding to the causal power, but if it remains insufficient for the event to occur across the sample, then logistic regression would consider

there a 0% chance of the event occurring in the presence of the moderator. Clearly contributory causes are not easily identified using logistic regression.

Whether an injury occurs depends on the complex interactions between moderating variables and therefore has many contributory causes. Whilst one variable may increase the probability of the event, another may mitigate its effect. As a result, the association between the moderating variable and injury is not identified despite the existence of an underlying causal mechanism. Observational studies appeal to Murphy's fallacious Law, that, because an event is expected to occur, it will occur. Or in this context, because a variable increases the risk of an event, the event will happen. The origin of these concerns arises from assuming persons who experience the event will possess a common cause. However, many causes can exist and interact with each other. Even if a common trait were found, this does not mean the full stratum of causes are identical with equal magnitude.

The immediate solution that comes to mind is multivariable analyses, but this too assumes individuals will experience an event from an identical combination of causes with similar causal effects. Whilst it is expected the effects will arise along the same causal path, the moderating variables that determine how that path is taken will differ, and it is improbable any two cases will be identical. For this reason, associations between moderating variables and effects cannot be repeated, or found at all, unless it alone is necessary and sufficient for the effect. Likewise, with each parameter added to the model, the greater the sample size needed to reach a sufficient power or precision to estimate the risk. Thus, multivariable analyses become more laborious by requiring additional variables to be measured across a larger sample size. The measurement of many variables is impractical, and some may not even be possible; a problem Pearl, Glymour and Jewell (2016) highlighted in simulated interventions on observational data. These statistics, both univariable and multivariable, do not inform the location of their effect along the causal path and cannot provide evidence of the underlying mechanism (Pearl, Glymour and Jewell 2016), which is useful for the development of interventions and analogous causes.

Nonetheless, some moderating variables will possess a more potent influence than others and it is possible these could be identified through observation because of their dominant causal power. For variables with a smaller influence this would not be the case. Using the specific case of hamstring injuries (the topic of this thesis) as an example, multiple variables have been associated with injury (Schuermans *et al.* 2017a; Duhig *et al.* 2016; Timmins, *et al.* 201) but it is expected others have been missed. Variables such as eccentric strength, which is repeatedly associated to injury (van Dyk *et al.* 2016; Timmins *et al.* 2016; Opar *et al.* 2015), might do so only because of its interactions between moderating variables such as muscle architecture and the neuromuscular system. Likewise, strength itself may have a direct moderating effect. Consequently, it is unknown what the causal power of those variables is. Observational methods demand substantial resources yet provide limited information and it is for this reason that alternative approaches should be considered.

Perhaps if a variable (X) is found to have no association with another (Y) in observational data, then its causal power on the corresponding mediating event is unlikely to be worthwhile, or is null, although it may increase the probability of Y. There are two reasons this is not the case. First, identifying moderating variables can build our understanding of the causal mechanism regardless of their causal power, and according to Bradford Hill's criteria of causation, understanding causal paths can help identify causes in analogous events (Hill 1965). Second, once moderating variables are identified and addressed through intervention, the cause of an event will fall upon the remaining moderating variables with the greatest probability to bring about Y. These variables would have been unidentifiable through observation because of the strength of the other moderating variables. Moderators that previously had a smaller role become the dominant cause of Y as others are removed and observational data would detect these variables once they become the dominant cause. It is expected these variables would have already been disregarded from earlier research. Moreover, when these variables should be investigated requires knowledge of the conditional probability of Y given the moderating variable X, which cannot be known unless the causal mechanism has already been identified and further research would be redundant. Observational

studies have unquestionably contributed to knowledge and allowed scientific progress, but the chance of erroneous inferences from observational data appears large.

A solution to these problems is experiments. It was stated that experimentation is not possible in epidemiology, as the event of interest cannot be induced. However, along any causal slide exists mediating events necessary for the event to occur. According to the logic of Modus Tollens, a moderating variable that increases the probability of a mediating event must also increase the probability of the event of interest. Likewise, mediating events are easier to identify through observation, as by necessity they must exist in all whom experience the event. In other words, If X then Y, then observing Y must mean X occurred at a prior instance. Whereas the presence of a moderating variable Z is not guaranteed in the presence of Y.

Surrogate markers are measurements that can be used to predict events, such as hamstring injury (Weintraub, Lüscher and Pocock 2015), but recent articles have discouraged their use (Weintraub, Lüscher and Pocock 2015; McNulty and Williams 2014; Grimes and Schulz 2005). A criticism of surrogate markers is that it does not always capture the net effect on the outcome (Weintraub, Lüscher and Pocock 2015). So, whilst a surrogate marker may improve from the intervention, other factors may become more severe and result in a net outcome that is damaging. This emphasises the need for well thought out interventions on a specified causal slide (path) to prevent confounding.

Likewise, a change in a surrogate marker does not always equate to a change in the effect (McNulty and Williams 2014; Grimes and Schulz 2005) but does not mean interventions that reduce the causal power without removing the event are not useful. For example, two interventions on their own may not reduce causal power to a level necessary to prevent the effect, but combined they may well do so. These viewpoints are from a medical perspective where focus is towards preventative interventions and observable outcomes. Epidemiology does not have this luxury, but surrogate markers offer the possibility to use ethical experimental designs and obtain robust evidence compared to observational outcomes. It is considered more useful to obtain reliable results of a surrogate marker than speculative results on the outcome of interest and declaring a variable non-

causal because the event did not manifest (or continued to manifest) would be inappropriate and potentially damaging.

More problematic is selecting the mediating event to represent as an outcome of interest (the surrogate marker) in experimentation. If the purpose is to identify variables that can be intervened on with the greatest success of reducing the undesirable event, then the mediating event with the greatest conditional probability should be chosen. That is, given you observe the prior mediating event (A), what is the probability you will observe the next mediating event (B), or P(B|A), without conditioning on any knowledge of Z (moderating variables). This will provide an indication of the moderating variables that have the largest causal power and therefore the largest potential for intervention. An alternative option is to choose the most proximal mediating event from the effect, in case there is a fork in the causal mechanism yet to be discovered that leads to the event via a backdoor path (Pearl, Glymour and Jewell 2016).

The experimental approach to investigate the chosen outcome must also meet a series of assumptions to make causal inferences (Hernán 2016). Randomised controlled trials are considered the gold standard because the randomisation process meets two key assumptions: The Positivity and Exchangeability assumption. Positivity requires all individuals to have a probability of belonging to either treatment or event group. Exchangeability ensures that participants are not assigned to a group based on an unrelated confounder. Both are met through randomisation alone. A third assumption that must be is Consistency, which ensures the effects observed under an intervention that sets X = x are consistent with the effect that would occur had nature set X = x. This prevents composite causes from influencing the effect. Consistency is often violated when the intervention is vague and multiple causes exist for a single intervention. For example, the causal effect of reducing obesity on blood pressure will depend on how obesity was reduced, such as diet and exercise (Hernán 2016).

Yet despite these assumptions, knowledge of the cause is limited to the intervention procedure itself rather than some abstraction of what the intervention represents. As empirical knowledge is limited

to the temporal and spatial observation of objects, causes and effects are assumed as the process between them is not known. It is possible the intervention is a common cause of the proposed cause and effect variables as per box b in figure 2. Here, the two variables would be associated as both respond to the intervention, but neither are causal towards one another.

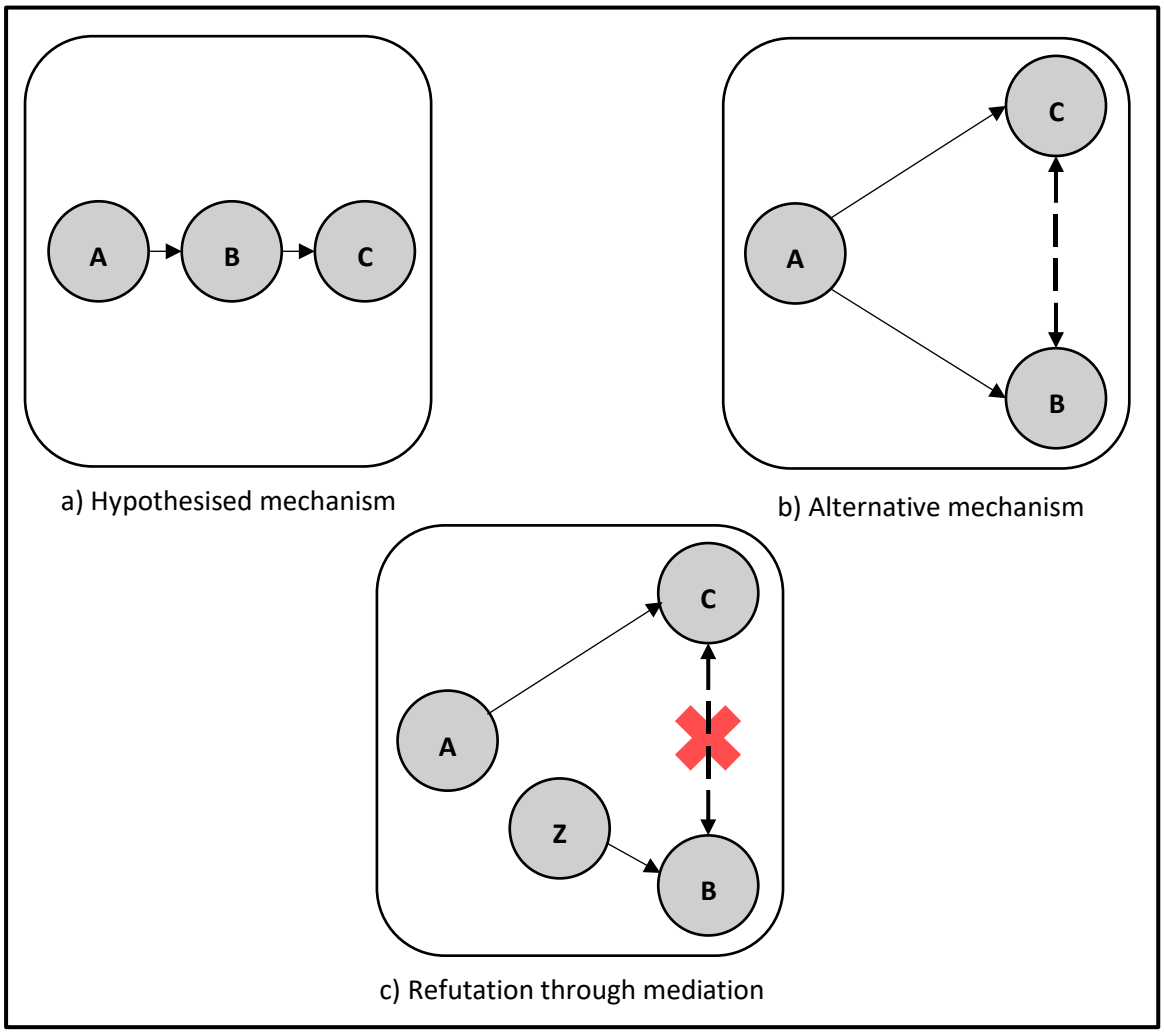

Figure 2: How tests of mediation can assist refutation of causal claims. **Note:** Solid lines represent causation; Dashed lines represent association.

Causal assumptions can be falsified by investigating mediating causes unique to the proposed causal path. Figure 2 depicts a hypothesised causal mechanism (box a) and an alternative mechanism (box b). The hypothesis suggests a causal association between A, B, and C, but the alternative mechanism is also a possibility. An attempt to falsify the association between A and C, and therefore increase our beliefs, can be done by intervening on a mediating event, called 'Z', along the proposed causal path (box c). By intervening on Z, all prior connections to A are absent and the association between B and C is absent; therefore, the causal effect of B on C is falsified. It is possible that Z could also

produce C through a backdoor mechanism, but as more mediators are examined, the less sample space (time) is available for these confounders to alter the outcome. The smaller the temporal interval between observations, the stronger the premise X causes Y. To determine the causal effect of a moderating variable, a change in the associating mediating event must be observed. Whether this is meaningful is a matter for expert reasoning rather than statistics as every context is different. However, once multiple moderating variables are identified, those with the greatest increase in probability or magnitude of the chosen mediating event should be intervened on, as these indicate a greater benefit through mitigation. Once sufficient variables have been mitigated or removed, the causal path becomes disrupted and the final event cannot occur.

1.4.2. Methodical considerations

To have evidence of causality the research question proposed by the thesis must be answered using methods that meet the three assumptions. Both exchangeability and positivity are met easily in experiments through randomised assignment or repeated measures designs with random recruitment. The consistency assumption requires core weakness to be defined to a context and that this process has no side-effects that would alter the causal effect you would expect to observe under natural circumstances. Determining a causal effect requires a specified context that would be expected to occur in nature. However, a cause must be manipulated to ascertain its effect. Therefore, baseline core strength is not an appropriate context. Soccer HSIs seem to increase towards the end of match play (Woods *et al.* 2004), so inducing a change in core strength through fatigue would align with these observations.

1.5 CONCLUSION

This thesis was originally tasked to identify whether core muscle weakness causes an increased risk of injury in soccer. In order to determine whether core muscle weakness causes increased injury risk, it is clear that the weakness must be induced experimentally to a degree equivalent with what occurs in soccer, and without affecting a variable indirectly influencing the risk of injury. Thus, the following

section will review the literature of hamstring strain injuries to conceive a conceptual mechanism of hamstring injury that specifies the variables involved.

SECTION 2:

DEVELOPING A MODEL OF THE INJURY MECHANISM AND THE POTENTIAL ROLE OF CORE MUSCLE WEAKNESS

2.1. THE EPIDEMIOLOGY OF HAMSTRING STRAIN INJURIES

2.1.2. Comparing injury incidence across activities

it is useful to compare HSI incidence across activities to better understand the potential cause of HSIs in soccer. In a broad review of hamstring injuries, the most common injuring activities were ball sports (44%) and running (24%; Kuske *et al.* 2016). Indeed, HSIs are prominent across sports but the absence of contact during incidence is a recurring finding (Ueblacker, Müller-Wohlfahrt and Ekstrand 2015; Dalton, Kerr and Dompier 2015) along with the frequency for injury to occur whilst sprinting or during manoeuvres associated with speed and agility (Fuller, Taylor and Raftery 2016; Dalton, Kerr and Dompier 2015; Ueblacker, Müller-Wohlfahrt and Ekstrand 2015; Edouard *et al.* 2014; Brooks *et al.* 2005; Orchard *et al.* 2002). Reflecting this observation, incidence in elite athletic events such as hurdles, sprinting, and endurance tasks is greater than in elite soccer (champions league) per hour of exposure (31.5/1000 hrs and 5.56/1000 hrs respectively; Ueblacker, Müller-Wohlfahrt and Ekstrand 2015; Edouard *et al.* 2014), which can be attributed to the fact athletic events consist of continuous high-speed running. Though endurance tasks are unlikely to include high-speed and agility tasks and suggests fatigue may be a contributing factor.

In baseball the injury rate is markedly lower (0.7 per 1000 exposures) and yet they remain the most common injury in the sport (Ahmad *et al.* 2014). A study of longer duration found the rate of HSIs to be greater (1.09–1.17 per 1000 exposures; Okoroha *et al.* 2019) but it is still dwarfed by other ball sports such as soccer. The principal cause of injury in baseball was running (Okoroha *et al.* 2019) as it is for athletic events, but the frequency of running is expected to be less than in sports such as soccer and explains the lower incidence rate. In rugby union, posterior thigh injuries are more frequent in backs than in forwards (13.2% and 5.1% respectively; Fuller, Taylor and Raftery 2016) and remains so at the community level (recreational and semi-professional rugby; Roberts *et al.* 2013). It is unlikely to be a coincidence that backs perform more sprints and high-speed manoeuvres than their forward positioned teammates (Cunningham *et al.* 2016). Specifically, Brooks and Kemp (2011) suggested blind-side flankers and fly-halves are most susceptible to HSIs but this was based on injury severity and not incidence alone. An analysis of proximal hamstring injuries identified high-

speed running-based sports as the most likely to induce injury (soccer [16%], sprinting [9%] and tennis [9%]; Irger *et al.* 2019). The high incidence during high-speed manoeuvres indicates there may be a task specific mechanism of injury.

2.1.3. Comparing injury incidence to exposure

In soccer, HSIs increase in the final third of each half (Woods *et al.* 2004). Similarly, in athletics, the 4 × 400 m sprint displayed a higher risk than the 4 x 100 m (Opar *et al.* 2013) — though incidence was not relative to exposure and the greater risk may arise from the additional 300 m of sprinting. Nonetheless, there is good evidence to suggest fatigue, and not increased exposure, increases the injury risk. The incidence rate for all thigh injuries in Qatari soccer is just 0.4/1000 hrs compared to 5.56/1000 hrs in the champions league, where exposure is greater (254 and 556 hrs respectively; (Ueblacker, Müller-Wohlfahrt and Ekstrand 2015; Eirale *et al.* 2013) and thus fatigue more likely. It is not surprising Ekstrand, Hägglund and Waldén (2011) identified a greater frequency of HSIs during the competitive season than the pre-season, where fixtures may be more congested. Fixture congestion has demonstrated a linear association with injury frequency (β: 0.52, CI: 0.11-0.93; (Bengtsson, Ekstrand and Hägglund 2013). However, a low R^2 value (0.005) suggests other factors are involved. Sahfiq, Masqood and Arshad (2016) proposed that age, an absence of a cool down, stretching, and a history of previous HSI, alongside fatigue are all risks for injury in community soccer. Though, this was based on frequency data alone and not inferential statistics, combined with only three months of observation. In addition, the injury diagnosis had a sensitivity and specificity of 0.55 and 0.30 so the sample of injured players may not represent the population with HSIs (Shafiq, Masqood and Arshad 2016).

An alternative explanation for the greater incidence in the competitive season than the pre-season is more competitive match play, which is expected to be more physically demanding and thus require more high-speed manoeuvres. In alignment, HSIs are greater in soccer matches than in training despite the absence of direct trauma in injury incidence (5.56/1000 hrs and 0.71/1000 hrs respectively; Ueblacker, Müller-Wohlfahrt and Ekstrand 2015). This finding has been observed over

many sports for both males and females (rate ratio: 2.05; Dalton, Kerr and Dompier 2015). Further to the argument against fatigue as a risk, community rugby had a greater incidence in the first quarter than the third quarter (1.9/1000 hrs and 1.1/1000 hrs respectively; Roberts *et al*. 2013), though a difference of 0.9 injuries per 1000 hrs and is unlikely to possess practical importance as it equates to one more injury every 750 matches. In Gaelic football, most HSIs occurred during training rather than matches, but injuries were not relative to exposure (O'Connor *et al.* 2016). If the frequency of training is assumed greater than competition, which it likely is, this would explain the greater incidence during training (21.7% and 11.5% respectively; O'Connor *et al.* 2016).

Overall, the evidence indicates more HSIs with fatigue, but this is not certain. Nonetheless, there are some associations between indicators of fatigue and injury. When the average high-speed running distance (≥ 24km/h) over four weeks is greater than the 2-year average, the risk of HSI increases (OR = 1.96 [CI = 1.54–2.51]; Duhig *et al.* 2016) but substantial α inflation exists for both variable selection (five variables) and the time frame for calculating the acute workload (four blocks), but the results of the models often report p values < 0.001, which are likely to fall below a corrected error rate. In contrast, HSIs were not associated with decreased rest between matches in the NFL (< 5 days; Lawrence, Comper and Hutchison 2016) but perhaps three- or four-days rest is sufficient for this sport. In runners, the more weekly miles completed the greater the HSI risk (OR = 1.11; p = 0.005; Wen, Puffer and Schmalzried 1997). Orchard *et al.* (2012) found seven or more interchanges (substitutions) for a player in the last three weeks were protective of injury (relative risk = 0.74), yet over 60 opposition interchanges in a single Australian football match increased the risk of injury (relative risk = 1.38). Using substitutions is consequently paradoxical in team sports as whilst it protects the substituted player, it puts the opposition at increased risk by maintaining intensity of match play. Considering this evidence, it's possible that some causes of HSIs may lurk in fatigued conditions and may not be visible at rest.

2.1.4. Observations of injury incidence

Hamstring strain injuries are prominent during water-skiing and appear due to rapid hip flexion whilst the knee is extended in the closed chain (Sallay *et al.* 1996; Blasier and Morawa 1990). Though rapid movement does not seem a requirement for injury, as injuries following a similar kinematic pattern (stretching and sagittal plane splits) but with small forces injured the proximal semimembranosus (SM) tendon (Askling *et al.* 2008; Askling *et al.* 2007b). In sprinting, where forces are higher and lengths are relatively smaller, injuries appear at the biceps femoris (BF; Askling *et al.* 2007a). Thus, the location of injury may be specific to the injuring scenario. In athletics, where running manoeuvres are common, the most commonly injured hamstring was also the BF long head (75.6%; Malliaropoulos *et al.* 2010) which is consistent with Askling *et al.* (2007a). Verrall *et al.* (2003) noted 81% of injuries were within the BF according to MRI, and those with a sudden onset occurred most frequently during running (65/68).

A retrospective review of 179 MRI and sonography images of HSIs in soccer, athletics, cricket, and water-skiing athletes identified the BF as the most commonly injured hamstring (80.5%), with 61.3% suffering musculo-tendon-junction (MTJ) injuries and the remaining occurring at the epimysium – though the distinction between them is not clear (Koulouris and Connell 2003). Precisely, the proximal and mid-portion were the most commonly injured sites of the BF (Koulouris and Connell 2003), and these findings were later mirrored in Australian footballers (Koulouris *et al.* 2007). In soccer specifically, the proportion injuries in the BF is lower but consistent with previous research as the most commonly injured hamstring (56.5% of 275 injuries) followed by the ST (24.4%; Crema *et al.* 2015). Injuries to the proximal BF MTJ (26.7%) was reported the most common followed by the distal MTJ (12%; Crema *et al.* 2015). In the ST, injuries were largely confided to the muscle belly (15.3%; Crema *et al.* 2015). Clearly, the BF MTJ is a vulnerable site for soccer players and as this site of injury is commonly associated with running tasks during injury incidence (Okoroha *et al.* 2019; Askling *et al.* 2007a; Verrall *et al.* 2003), it further suggests running as the prominent cause of injury during soccer. Understanding the mechanics of the biceps femoris during high-speed running manoeuvres would provide a more in-depth insight into the mechanism of injury in soccer and begin

to detail the causal pathway for hamstring strain injury along with the moderating and mediating variables.

2.2. STRESS AS A CAUSE OF STRAIN INJURIES

2.2.1. introduction

Two mechanisms of hamstring injury appear to exist and are referred to as stretching and high-speed (forceful; Askling, Saartok and Thorstensson 2006). This classification arises from observations in dancers where HSIs occur at low forces but long absolute lengths (Askling *et al.* 2007b) and injuries in sprinters that occur at short absolute lengths but with high forces (Askling *et al.* 2007a). Thus, these two posited mechanisms can be reduced to either excessive strain (change in length proportional to resting length) or excessive force. It's possible the relative strain is similar between these two groups, because sprinters might relatively short hamstrings, but the mechanism (excessive strain or excessive force) also seems to determine the muscle susceptible to injury. The SM is prone to injury under excessive strain (Askling *et al.* 2007b) whereas the BF is at risk in forceful scenarios, such as sprinting (Askling *et al.* 2007a). This suggests a unique quality exists dependent on the mechanism that renders each muscle susceptible to injury.

However, the notion of two separate mechanisms for strain injury is at odds with the experimental research in myofibrils, which suggests strain is the principal cause of injury (Patel *et al.* 2004; Lieber and Fridén 2002). In accordance, simulation studies have found the biceps femoris is most lengthened whilst sprinting, appearing to explain its susceptibility (Schache, A. *et al.* 2012; Chumanov, Heiderscheit and Thelen 2011; Chumanov, Heiderscheit and Thelen 2007; Thelen *et al.* 2005a). Yet, if length was the determiner of injury, then injuries during passive stretching would be a common occurrence. This is not the case as evidenced by stretching interventions that do not report injury due to the act of hamstring stretching (Arnason *et al.* 2007; Brooks *et al.* 2006; Nelson and Bandy 2004). Whilst it could be the case that the longer absolute strains in dancers achieve the same relative strains in spritners, due to differences in flexibility, it seems unlikely to be the case as flexibility is better represented by the nervous system than the muscle structure. Krabak *et al.* (2001) found flexibility is affected by anaesthetic, and foam rolling can alter contralateral ROM (Killen, Zelizney and Ye 2019). Furthermore, although the biceps femoris is most lengthened during

sprinting, its absolute length is small. At larger absolute strains the semimembranosus is more commonly injured (Askling *et al.* 2007b). Another variable must exist to explain this discrepancy.

2.2.2. The role of myofiber strain in injury mechanics

The notion of strain causing injury rather than force arises from a seminal study in myofibrils that compared strain magnitudes of 12.5% to 25% with either high or low levels of force, achieved by manipulating the delay between onset and strain (Lieber and Fridén 2002). They found that regardless of force, damage was always greater in groups with larger strains, yet similar between muscles exposed to different forces (Lieber and Fridén 2002). However, according to Hooke's Law (Eq.1) the spring constant (−*k*) means force (*F*) is proportional to strain (*s*). Thus, the high strain group intended to have low force could in fact have had greater force than the low strain group intended for high force. This would confound any attempt to attribute injury to strain or force.

$$F = -k \times s \tag{1}$$

It was reported that force was 40% greater in myofibrils in the high force group compared to the low force group but was only reported for myofibrils with a large strain magnitude. No values of the forces are given for the low strain group. Through Eq. 1, it is possible to estimate the forces in myofibrils of different strains and intended forces from Lieber and Fridén (2002). Therefore, the aim of this investigation was to estimate the force in myofibrils of different strains and forces (onset times). It is hypothesised that force in the high strain/low force group will be greater than the low strain/high force group.

2.2.2.1. Method

The force produced by the low force group with large strain (25%) was estimated using WebPlotDigitizer (Rohatgi 2019) and figure 2A of Lieber and Fridén (2002), and the force was converted from grams to newtons using a ratio of 102:1. This was equal to 14 N. Nominal strain is given at 55 mm, and it is stated by Lieber and Fridén that forces in the high force group with 25% strain is 1.4 times greater than the low force group with 25% strain. Thus, the myofibril forces for each condition can be derived using the calculations provided in table 1. Where spring constants for

the early stretch (k_{ES}) and late stretch (k_{LS}) groups of 12.5% strain are estimated using Hooke's Law by dividing the force by absolute strain in the 25% strain group. The results of these calculations are displayed in table 2.

Table 1: Calculations for estimating myofibril forces

Strain		Force	
Relative Strain (%)	Absolute strain (mm)	Low force (N)	High force (N)
25	$S_{25} = 55 \times 0.250$	A = 14	$B = A \times 1.40$
12.5	$S_{12.5} = 55 \times 0.125$	$C = k_{ES} \times S_{12.5}$	$D = k_{LS} \times S_{12.5}$

2.2.2.2. Results

Table 2 shows myofibril forces are greater in the high strain/low force group compared to the low strain/high force group (14N and 10N respectively). The mean difference in force between timings is ~4.5 N, whereas the mean difference in force between strains is ~8.5 N.

Table 2: Estimates of myofibril force in each condition

Strain		Force	
Relative Strain (%)	Absolute strain (mm)	Low force (N)	High force (N)
25	13.75	14	20
12.5	6.875	7	10

2.2.2.3. Discussion

Frieden and Lieber (2002) concluded strain is the principal cause of injury based on a significant effect in the strain condition but not a significant effect in the onset time condition on post exercise force reductions (a marker of muscle damage). However, the forces within these groups were not reported. The results of this analysis show that at high strains, the group intended to represent low force had 4 N greater force than the group intended for higher force but at low strains. The suggestion that strain is responsible for injury rather than force could be misleading, as strains leads

to greater forces. The additional 4 N of force from greater strains is considerable given the peak tetanic tension in myofibrils is ~13 N, and suggests that lengthening, on average, increased force by 31% of its peak force than when manipulating the force production through onset time.

As strain created greater forces, then it may seem that Lieber and Fridén (2002) were correct in their conclusion that lengthening causes injury. Whilst we agree with Lieber and Fridén (2002) that greater strains lead to greater muscle damage, stating it is not the result of high force is incorrect and not supported by the data. Instead, lengthening is a means of achieving greater forces. Under maximal conditions, and homogenous myofibrils, such as those used by Frieden and Lieber (2002), the larger strains will cause greater forces and thus injury. This relationship does not hold between heterogenous muscles because of differences in passive tension and active force production. Therefore, identifying force as the cause of injury is a subtle but important difference when comparing muscles.

Yet, these conclusions may still be limited to myofibrils, as the largest contributor to force when sprinting is the SM, and not the commonly injured BF (Schache *et al.* 2012; Chumanov, Heiderscheit and Thelen 2011; Chumanov, Heiderscheit and Thelen 2007). Sprint simulations have also combined force and strain by calculating Work to explain the BF's vulnerability, but this too has failed (Chumanov, Heiderscheit and Thelen 2011). Despite force seemingly causing injury in myofibrils, the current outcomes from sprint simulations do not identify the vulnerability of the biceps femoris in this common injury inducing movement. A new perspective is warranted to converge the observed data from simulations to that in myofibril experiments.

2.2.3 Estimates of stress between the hamstring muscles

It is well established in Newtonian mechanics that materials fracture under excessive tensile stress (Ashby and Jones 2012). Stress is the measure of the internal force acting in a localised area (Eq. 2). From the perspective that muscle is a biological material, the cause of fracture (injury) should not differ. Thus, stress is likely to be the principle cause of injury. In contrast to reported outcomes, this requires not only the forces to be considered but the area of the muscle too. In myofiber research

the CSA of homogenous myofibrils is expected to be similar and therefore differences in force would be proportional to stress, such that the force induced by strain would appear as the determining factor. As muscles differ in CSA, this does not hold true.

$$\sigma = \frac{F}{A} \quad (2)$$

Stress as the cause of injury can explain why the MTJ is a prevalent location for injury as the area lessens as the muscle tapers to the free tendon (Storey *et al.* 2016). This has been alluded to by Storey *et al*. (2016) but an explicit investigation has not been performed. Earlier studies have investigated the aponeurosis size in relation to eccentric strength ($r = 0.24$; $p > 0.2$) but not regarding injury incidence (Evangelidis *et al.* 2015).

The SM is the largest producer of force whilst sprinting, but a large CSA would reduce the stress. Conversely, a small CSA in BF would increase its stress and propensity for injury. The aim of this study was to identify whether stress is greatest in the biceps femoris by approximating the stress at each hamstring's MTJ during sprinting using previous simulation and morphology data. The hypothesis was that MTJ stress will be greatest in the BF.

2.2.3.1. Method

The peak force produced by each hamstring muscle when sprinting was obtained from simulation data and combined with morphological data to estimate the stress at the MTJ for each hamstring muscle. The area of the MTJ for each hamstring muscle was obtained from Storey *et al.* (2016). Sprint simulations were identified through prior knowledge and confirmed via a PubMed search using the terms sprint*, simulation, and hamstring connected with the 'AND' Boolean operator. To be included, simulation studies had to identify the peak force for all three biarticular hamstrings (SM, semitendinosus [ST], and BF) whilst performing high-speed sprinting. The MTJ area data was obtained from different participants to those used in simulations, which may reduce the validity of these estimates, however, aponeurosis area is not related to muscle size or area (Evangelidis *et al.* 2015) and so the average MTJ areas from Storey *et al.* (2016) are a fair approximation of the participants used in the simulation studies.

Using R statistical software, a random effects meta-analysis (Viechtbauer 2010) was conducted to account for between study variability using the peak force from the three simulations to derive an estimate of the 95% CI for peak force in each muscle. Likewise, a two-sided 95% CI for each MTJ CSA was estimated from the data provided by Storey *et al*. (2016). The point estimates of the intervals (lower bound, mean, and upper bound) for both force and MTJ area were used to derive a corresponding 95% CI estimate of the stress in each muscle following Eq. 2.

2.2.3.2. Results

Three studies were retrieved for analysis of hamstring force whilst sprinting (Schache, *et al.* 2012; Chumanov, Heiderscheit and Thelen 2011; Chumanov, Heiderscheit and Thelen 2007) with a combined total of 38 participants (28 males and 10 females; Age: 24 years; stature: 177 cm; mass: 73 kg). For the morphology data, five male cadavers (10 limbs) were used (mean age: 75 years [no dispersion reported]; Storey *et al*. 2016). Table 3 includes the random effects confidence interval for peak force among the three simulation studies and the confidence interval for MTJ area for each muscle. On average, the biceps femoris experiences 4.24 $N \cdot cm^2$ more stress than the SM and 7.36 $N \cdot cm^2$ more than the ST. The variability in the biceps femoris stress estimate is also greater than the SM and ST. This is highlighted at the upper bounds of the stress estimate where the difference between BF and SM increased to 9.73 $N \cdot cm^2$.

Table 3: Estimates of 95% confidence intervals for peak force, MTJ area, and MTJ stress.

	Muscle	Lower bound	Mean	Upper bound
Peak force* (N)	BF	861	1408.4	1960.7
	SM	1470	2289	3108
	ST	340.2	450.1	559.3
MTJ Area (cm^2)	BF	39.4	45.0	50.6
	SM	62.1	84.6	107.1
	ST	16.2	18.8	21.4
MTJ Stress ($N \cdot cm^2$)	BF	21.85	31.30	38.75
	SM	23.67	27.06	29.02
	ST	21.00	23.94	26.14

Note: *Absolute peak force has been calculated for a 70 kg person.

2.2.3.3. Discussion

The aim of this investigation was to estimate whether stress could identify the BF vulnerability to injury during high-speed running. The findings presented here show the BF experiences the greatest MTJ stress. Prior to this study, strain was the only outcome to identify the biceps femoris vulnerability, but it is implausible for strain alone to be responsible for injury. Thus, stress is the first variable to align with both simulated and myofibril research. It is expected that the stress estimates for the BF may be greater in reality. Chumanov's *et al.* (2011) simulation found the BF had a small force contribution which seems to occur from the notably low BF excitation during simulation that does not align with electromyography (EMG) data (Chumanov, Heiderscheit and Thelen 2011). Thus, the increased stress, and thus injury risk, in the BF might be clearer that found here.

Stress as the cause of injury has fundamental implications towards our understanding of injury mechanics. Previously, lengthening was believed to be the fundamental cause of injury (Chumanov, Heiderscheit and Thelen 2011; Lieber and Fridén 2002), but as demonstrated, lengthening likely causes injury because it results in greater forces. The notion of stress rather than strain as the muscle damaging factor aligns with earlier findings that showed muscle damage after shortening muscle actions (Gibala *et al.* 1995). A phenomenon not possible according to the lengthening model. Nonetheless, strain may have an additive effect to injury risk separate from the increased force. The volume of the MTJ does not change during lengthening, therefore an increase in strain would result in a decrease in MTJ area and a subsequent increase in stress (Ashby and Jones 2012). Although not the principal cause, the greater strain in the BF whilst sprinting may compound its vulnerability to injury here. Estimates of maximum hamstring muscle CSA show the SM undergoes the smallest reduction (2.3%) compared to the BF which reduces the most (8.6%; Nakaizumi *et al.* 2019). Therefore, stress at peak lengths for the biceps femoris are expected to be considerably larger than estimated here, as MTJ measurement used in this analysis were measured ex vivo and not whilst lengthened.

More critical implications exist for our understanding of injury prevention. The maximum force in a local area that a material can withstand could be the principal factor in preventing injury. Understanding the factors that determine this threshold would be of great value for optimising injury prevention programmes and screening. For example, the protective role of structures binding actin to the extracellular matrix has been shown in mice studies. Mice over-expressing the α7BX2 integrin (a common isoform of a muscular integrin) display reduced membrane damage after downhill running ($p < 0.05$) suggesting the increase in integrin and actin to laminin connections increase structural soundness (Boppart, Burkin and Kaufman 2006). Adding micro dystrophin to mdx mice also reduces muscle damage (Banks, Combs and Chamberlain 2008). Likewise, the ACTN3 R577X polymorphism (alpha actinin 3 which binds to z discs) is associated with greater shear modulus in the hamstrings, suggesting larger stress and thus risk (Miyamoto *et al.* 2018). Whilst this polymorphism was not associated to injury, the stress value for the modulus was calculated at the muscle belly and not the MTJ (Miyamoto *et al.* 2018).

The inability to produce eccentric force is associated with hamstring injury (Lee *et al.* 2018; Timmins *et al.* 2016) and accordingly strength training has shown to reduce hamstring injury incidence (van der Horst *et al.* 2015). Yet, if stress is the principal factor causing injury, then increasing the capacity to produce force should increase the risk of injury. One explanation is that strength training increases collagen in the MTJ, likely to manage to the new capacity to produce force (Jakobsen *et al.* 2017). In addition, eccentric strengthening may be important for reducing fascicle strain (Franchi, Reeves and Narici 2017) and thus reducing the passive force component from strain.

There are inevitable limitations with the approach used in this investigation. Using the peak force data from simulations for comparison assumes that the force distribution is equivalent within each muscle. This is unlikely considering fascicle strain appears non-linear (Fiorentino *et al.* 2014), but lengthening is concentrated towards the MTJ adding validity to use of peak force in MTJ stress estimates.

2.2.4. Conclusion

Until now, simulation studies have failed to identify a cause of injury that aligns with the understanding of injury in myofibril research. Using the data of simulations, this study has shown that stress is greatest for the BF whilst sprinting and can explain its susceptibility to injury, particularly at the MTJ. Though strain may still be useful measure for estimating injury risk but only if all other factors are constant (i.e. repeated measures designs with equivalent active muscle forces) as it would indicate reductions in CSA.

2.3. THE BIOMECHANICS OF THE HAMSTRINGS

2.3.1. Introduction

The previous text detailed the cause of injury at the myofiber level. Now, the cause of injury at the whole-body level will be considered. Following this, it will be possible to conceptualise the causal pathway of HSIs during high-speed running and subsequently identify how and where in the causal pathway core muscle weakness could increase the probability of HSI, if at all.

The earliest research available on hamstring injuries is a case study that identified the forcible flexing of the hip whilst the knee was extended as a cause of hamstring ruptures (Overton and England 1954). Slocum and James' (1968) hypothesis soon followed suggesting HSIs originate in the swing phase of running, where an eccentric hamstring action is required to decelerate knee extension when the hip is also flexed. Worth (1969) offered a similar postulate that HSIs arise from the need for concentric hamstring action to propel the body whilst suddenly flexing at the hip, causing eccentric contraction and muscle tearing.

At the time, views on strain injury mechanisms were primarily informed by expert opinion (Thiart 1973; Gilcreest 1933; Gilcreest 1925) rather than empirical research, yet for the hamstrings, forced lengthening had already been acknowledged as the likely cause (Worth 1969; Overton and England 1954). This mechanism was consistent with previous observations of muscle ruptures that occurred because of 'sudden pulls or jerks' 'when the muscle is in strong contraction' (Gilcreest 1925). Today, this can be interpreted as eccentric actions. Despite an apparent understanding of the underlying injury mechanism, HSIs remain prominent (Ekstrand, Jan, Waldén and Hägglund 2016), which suggests our current understanding is limited and effective intervention is needed. Mendiguchia, Alentorn-Geli and Brughelli (2012) expressed this perspective in their editorial entitled 'Hamstring strain injuries: are we heading in the right direction?'.

2.3.2. Hamstring mechanics during high-speed running

It was established in section 2.1. that HSIs tend to occur during high-speed manoeuvres such as sprinting. Specifically, it has been shown that these injuries are likely to occur during eccentric contractions because of the large stresses that can occur. The question remains, however, as to how such large stress arises in the hamstrings during high-speed running or sprinting. The following text seeks to identify the actions of the hamstring during high-speed running to better understand how they might contribute to stress.

Hamstring lengths peak during the swing phase of running, where the knee extends after peak hip flexion (Thelen *et al.* 2005a), and the BF strains more than the ST and SM (Wan *et al.* 2017; Thelen *et al.* 2005a). This aligns with the BF as the most commonly injured muscle in soccer (Crema *et al.* 2015). The terminal swing phase also coincides with peak force in the BF and SM adding to the propensity for hamstring injury during swing (Chumanov, Heiderscheit and Thelen 2011). More specific analyses in swing phase mechanics highlight the close temporal relationship between peak lengths and peak forces in the hamstring muscles; In the ST, the difference between peak force and peak strain is ~5–~9% of the gait cycle (Schache *et al.* 2012; Chumanov, Heiderscheit and Thelen 2007), but in the BF and SM this difference is less at ~1–~3% of the gait cycle (Nagano *et al.* 2014; Schache. *et al.* 2012; Chumanov, Heiderscheit and Thelen 2007; Thelen *et al.* 2005a).

These findings are replicated in overground sprinting too, with Higashihara *et al*. (2016), Ono *et al.* (2015) and Yu *et al.* (2008) all reporting close temporal relations between peak excitation or force and peak strain, however, these studies also report the medial hamstrings (either SM or ST) to be strained more than the BF. Yu *et al.* (2008) used absolute lengths rather than strain, which in inappropriate, but Ono *et al*. (2015) and Higashihara *et al.* (2016) used strain and the SM and ST remained lengthened more than the BF. The reason for this is not entirely clear but the overarching result for this body of research is that for all hamstring muscles, they undergo the greatest strain and forces during swing. Indeed, the most strained muscle is not necessarily the most at risk of injury according to a stress-based model of injury. Ono *et al.* (2015) acknowledged stress a cause of injury and combined EMG and strain data to create a 'tensile force index' indicative of the muscle

stress for each hamstring muscle, where tensile force = strain x EMG amplitude. The results showed the tensile force index peaked just prior to foot strike (terminal swing) for all hamstring muscles but was greatest for the ST. This is surprising as the ST represents just 24.4% of HSIs in soccer (Crema *et al.* 2015). This is the result of using EMG amplitude to estimate force, which is invalid and only indicates muscle excitation, not absolute force (Vigotsky *et al.* 2018). Furthermore, Fiorentino *et al.* (2014) showed the BF lengthens non-uniformly, therefore whole muscle strain would not represent the areas within the muscle with greatest strain. Whilst informative of the more hazardous portions of gait for the hamstring muscles as a whole, inferences regarding the individual hamstring muscle most at risk are misleading.

It is worth noting that the second peak in the tensile force index occurred during stance, just subsequent to touch down (Ono *et al.* 2015). This seems largely due to the peak in muscle excitation along with an extended knee joint (Ono *et al.* 2015). It is plausible that if there were any changes here that increased lengthening, then the stress may well exceed that in the swing phase. Indeed, it's important to recognise that evidence of hamstring kinetics and kinematics is collected during trials without the incidence of injury and the mechanism may well differ during running that consequences in HSI. Orchard (2012) proposed HSIs occur during stance because of the large hip flexion and knee extension torques that must be opposed. It's feasible that the inability to maintain typical kinematics due to external forces, such as the hip and knee torques, could lead to further lengthening and injury during stance, or that changes occur during stance that further increase the risk when entering the swing phase. In summary, the most hazardous portion of gait appears to be the terminal swing phase leading into the early stance phase in normal conditions. Though the most hazardous portion may differ in injuring runs. The following section will now consider the underlying causes of the large hamstring strain and forces during terminal swing and early stance phase of gait.

2.3.3. Causes of hamstring stress during high-speed running

If stress is the principal cause of muscle injury, then it's important to consider what factors contribute to hamstring lengthening and force production. The large hamstring excitation and length at stance appears to be in response to the anterior GRFs that create a knee extension and hip flexion moment, which must be opposed by the knee flexors and hip extensors (i.e. the hamstrings; Sun *et al.* 2015). The GRF acts mostly at the hip (Zhong *et al.* 2017), thus the inability to maintain hip extension and prevent hip flexion could be a source of additional lengthening and injury. Yet, evidence has failed to identify an association between hip extensor strength (Tokutake *et al.* 2018; Sugiura *et al.* 2008), gluteus maximus activity and volume (Franettovich Smith *et al.* 2016) and injury. Sugiura *et al.* (2008) reported a reduction in hip extensor strength between injured and healthy limbs in a prospective study, which is indicative of some relationship, but did not identify the same association between injured participants and controls adding some doubt that insufficient hip extensor strength leads to lengthening. In a kinematic study, Schuermans *et al.* (2017a) identified lower gluteus maximus activity during the initial swing phase in prospectively injured subjects so it may not be a lack of strength but a lack of co-ordination between the hip extensors that increases injury risk. Hamstring peak force is 229% greater than the gluteus maximus peak force during stance (3.19 $N \cdot kg^{-1}$ and 1.39 $N \cdot kg^{-1}$ respectively; Nagano *et al.* 2014) so it would seem there is potential for the gluteus maximus to reduce hamstring demands.

During the swing phase of running, the hip flexes due to muscular torque from the hip flexors (initiated by the iliacus and continued by the rectus femoris; Nagano *et al.* 2014), which is followed by angular acceleration of the shank, extending the knee (Zhong *et al.* 2017; Sun *et al.* 2015). An increase in acceleration of knee extension would offer some explanation for the increased hamstring activity in greater running speeds (Chumanov, Heiderscheit and Thelen 2011).

Prospective studies associating eccentric knee flexion weakness with injury risk (Timmins *et al.* 2016; Opar *et al.* 2015) and knee flexor eccentric training reducing HSIs (van der Horst *et al.* 2015; Petersen *et al.* 2011) suggest an improved ability to decelerate knee extension during swing, and thus limit lengthening, might lower stress and risk of injury. Though the act of strengthening or

being strong might reflect other qualities that lower the relative muscle strain such as longer fascicles (Timmins *et al.* 2016) or increased tendon stiffness (Thelen *et al.* 2005b). Indeed, an association between lower hamstring eccentric strength and injury is not consistently found (van Dyk *et al.* 2017), and eccentrically strong athletes are still susceptible to HSIs (Bourne *et al.* 2015). This indicates that muscle stress might arise from other sources than simply the strain from knee extension. An increase in hamstring strain might arise from weak hip extensors allowing greater hip flexion, but the majority of evidence suggests this might not be the case (Tokutake *et al.* 2018; Franettovich Smith *et al.* 2016; Sugiura *et al.* 2008). Factors such as force sharing between the hamstring muscles (Schuermans *et al.* 2014) and BF fascicle lengths (Timmins *et al.* 2016) may have a role too, but the potential for core weakness to increase lengthening via the pelvis (i.e. anterior tilt/hip flexion) is certainly an interesting prospect which is yet to be investigated using empirical observation and presents a plausible mechanism for core weakness to increase injury risk.

2.3.4. Causes of hamstring stress during fatigued high-speed running

Epidemiological evidence suggests a possible mechanism of fatigue in injury incidence (Woods *et al.* 2004) so it's important to consider whether the mechanism of injury changes under these conditions. Research has previously shown the strategy to generate horizontal force shifts from hamstring dominance to gluteus maximus dominance (Edouard *et al.* 2018; Morin *et al.* 2015) which might explain why hip extension weakness is not reduced in prospectively injured sprinters, as fatigue is less likely to occur in this sample.

The most enlightening evidence of fatigue's effect is from Small *et al.* (2009) who examined sprint kinematics after a 90-minute soccer simulation and found a decrease in peak knee extension and peak hip flexion. The reduction in peak knee extension angle is surprising as shank velocity increased, suggesting a reduced ability to decelerate the knee extension and thus result in greater lengthening (Small *et al.* 2010; Small *et al.* 2009). One possibility is afferent feedback increasing the perception of stiffness at relatively shorter lengths as indicated by a reduced hamstring ROM after

simulated soccer (Small 2008). Furthermore, whilst a reduction in peak hip flexion was reported after 90 minutes of soccer simulation, an increase in maximum anterior pelvic tilt was observed (Small *et al.* 2009), which might increase strain in the proximal fibres and align with the proximal MTJ as the most injured site on the BF (Crema *et al.* 2015). In summary, it seems the increased injury risk from fatigue arises from greater anterior pelvic tilt and not an increase in lengthening at the knee. The cause of this is unclear. It could be that the gluteus maximus becomes fatigued as it compensates for hamstring weakness contributing to further anterior pelvic tilt, but similarly the core muscles might become fatigued and alter kinematics such that the pelvis becomes more anteriorly tilted too.

2.3.5. Summary of the injury model

The previous literature detailed hamstring actions during sprinting, a common activity during HSI incidence, and identified the potential sources of hamstring stress (i.e. causes of hamstring lengthening and increased force). In turn, a set of possible causes for each source of stress has been speculated to offer a conceptual framework of the injury mechanism from which future research could be designed. It is hoped that by providing this framework, the appropriate measures can be made to control for confounding variables known at the time and have clear outcomes that can be used to infer an increased injury risk.

According to this model, presented in figure 3, the root cause of injury can be attributed to an external force that leads to knee extension or hip flexion, or both. Thus, the first line of defence might be to remove those external forces. In most cases this presents an unpractical solution as these forces are often important for performance. For example, horizontal force is associated with sprint speed (Morin, Edouard and Samozino 2011) and to reduce this force would reduce performance. From a practical perspective, the first line of defence for hamstring injuries should be to prevent excessive lengthening, either through increasing hamstring force or by increasing force in synergist muscles such as the hip extensors. The role of synergistic muscles such as the hip

extensors may be even more important under fatiguing conditions and additional lengthening may occur via an increase in anterior pelvic tilt. Thus, increasing or maintaining force in muscles associated with the pelvis (e.g. core muscles) might be useful but evidence of this is scarce. Lengthening could also be reduced at the sarcomere by altering the hamstring muscle architecture, such as fascicle lengths. Following this, damage will occur depending on the structure and integrity of the sarcomere itself (Boppart, Burkin and Kaufman 2006) and if excessive, induce damage at a macroscopic level.

Preventing excessive lengthening is considered the first protective barrier to injury according to this model. Research has investigated the protective benefits of stronger hamstrings (van der Horst *et al.* 2015; Petersen *et al.* 2011) and the potential for hip extensor weakness to increase injury risk (Sugiura *et al*. 2008) but the muscles that might be responsible for increasing anterior tilt are yet to be identified (Schuermans *et al.* 2017b). Thus, the potential for core muscles to increase injury risk via an increase in anterior pelvic tilt during sprinting is certainly worthy of consideration and the following text will consider the current evidence indicating a link between core strength and HSIs.

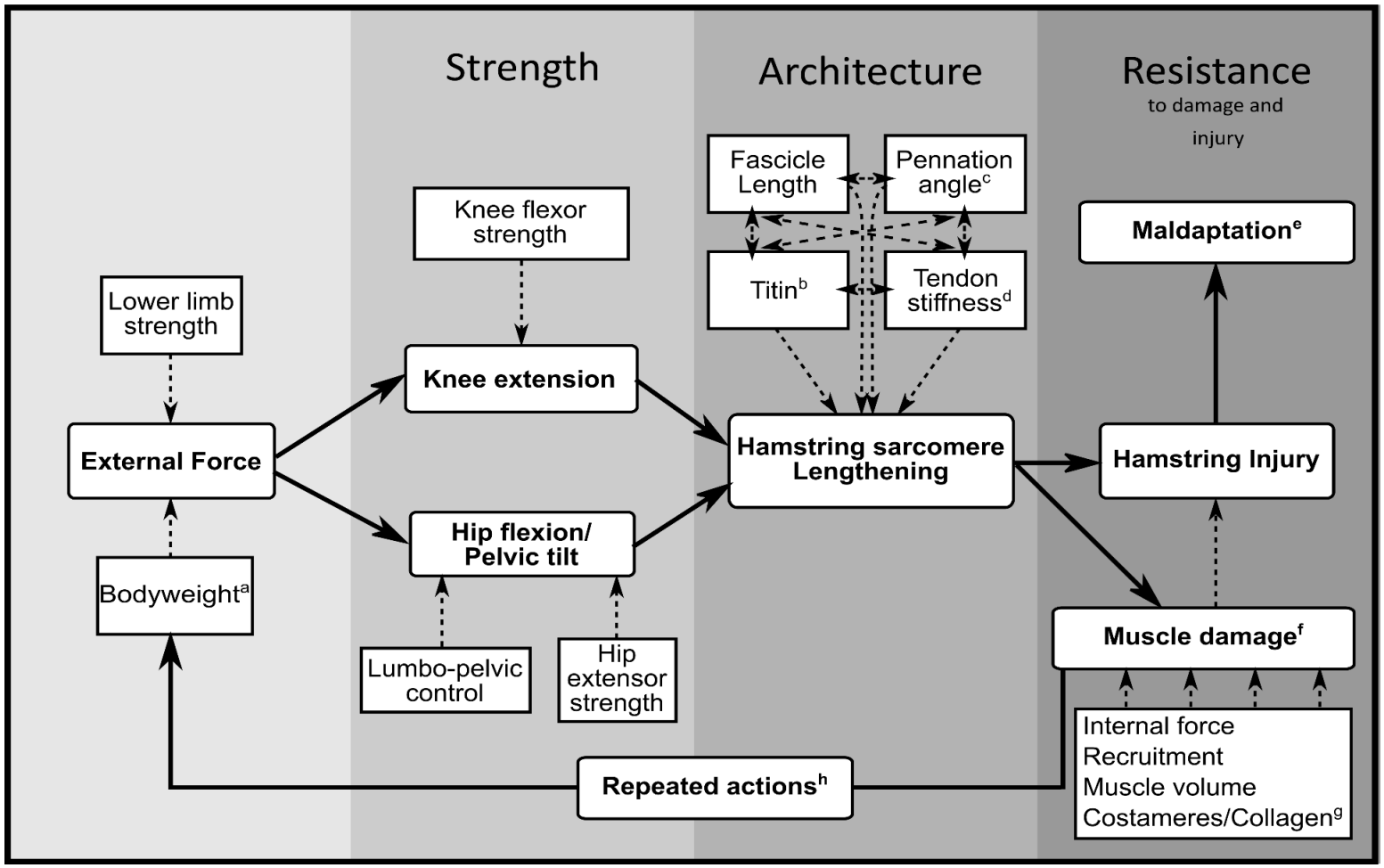

Figure 3: A model of the HSI injury mechanism. **Note:** Solid lines represent mediating variables. Dashed lines show moderating variables. [a]Hansen *et al.* (2017); [b]Perrin, Nosaka and Steele (2017); [c]Timmins *et al.* (2016); [d]Thelen *et al.* (2005b); [e]Green *et al.* (2020); [f]Larruskain *et al.* (2018b); [g]Boppart, Burkin and Kaufman (2006) and Banks, Combs and Chamberlain (2008); [h]Green *et al.* (2020)

2.4 THE POTENTIAL FOR CORE MUSCLE WEAKNESS TO INCREASE HAMSTRING INJURY RISK

2.4.1. Evidence associating the core muscles with hamstring injuries

Schuermans *et al.* (2017a) identified increased anterior pelvic tilt at late back swing (early front swing on the contralateral limb) in prospectively injured athletes which aligns with Daly (2017) in a cross-sectional study of healthy controls and previously hamstring injured participants. Schuermans (2017a) also observed an increase in frontal flexion of the thorax towards the swing limb at late swing. It's unclear how flexion of the thorax in the frontal plane increases injury risk but its possible thorax flexion and anterior pelvic tilt have a common cause, such as weakness in a particular muscle. Schuermans (2017a) also compared kinematics to those with a previous hamstring injury and found no difference compared to healthy controls (n = 30 injuries; Schuermans 2017a). This is surprising as a cause must precede effect. Accordingly, those with previous injuries must also have deviant kinematics — assuming they are a necessary condition for injury. It's possible the deviating kinematics were removed through a direct effect of injury, but this is unclear.

Schuermans *et al.* (2017b), as part of their prospective study comparing kinematics, also compared trunk muscle excitation (a single vector including the internal and external obliques and lumbar and thoracic extensors) between injured and uninjured participants during sprinting and found reduced excitation at the late backswing (contralateral terminal swing) in injured subjects, which is synchronous with the increase in anterior pelvic tilt and just prior to the thoracic frontal flexion identified in their similar study Schuermans *et al*. (2017a). Post-Hoc analyses were unable to identify the specific trunk muscle responsible but given the temporal proximity to the increased anterior pelvic tilt it would seem likely to be a muscle with pelvic attachments (i.e. obliques or lumbar extensors and not the thoracic extensors, though it's important to note Schuermans *et al.* (2017b) also identified lower gluteus muscle activity during early swing in prospectively injured subjects but this was not synchronous with the increase in anterior pelvic tilt. Though this does not mean it did not contribute

either. Bonte *et al*. (2015) in their Masters Thesis found previously hamstring injured participants displayed lower lumbar extensor excitation (96% MVC) compared to controls (192% MVC) during swing (stance not measured) and no difference in oblique excitation, which would indicate the erector spinae might be the muscle responsible for Schuermans findings (2017a). Bonte *et al.* (2015) also found the thorax was more flexed relative to the pelvis at touch down by 6.3° in injured athletes. Given the erector spinae prevents flexion of the spine and trunk (Thorstensson *et al.* 1982), this indicates the lower EMG activity reflected insufficient erector spinae force. In contrast, Dai Sugimoto *et al*. (2019) found injured subjects possess a more upright trunk when running but was observed at slow speeds (6.44–8.05 km/h) and although Bonte *et al.* (2015) does not state the speeds achieved in their findings, they were recorded at 15–20 m of a sprint where speeds are expected to be faster. In fact, a more upright trunk during slower speeds may be a compensatory mechanism to prevent excessive lean during faster speeds.

Insufficient erector spinae activity as the cause of injury is incompatible with simulation work from Chumanov, Heiderscheit and Thelen (2007) that showed hamstring lengthening is greater when lumbar extensor force is increased during the swing phase, likely because its actions produce anterior pelvic tilt. The reason for the discrepancy might be related to a differing function of the lumbar extensors during the swing and stance phase. During stance the erector spinae might act to keep the trunk upright as indicated by an increase in forward lean at touch down by Bonte *et al.* (2015), possibly preventing hip flexion as a result of anterior pelvic tilt, and in turn hamstring lengthening. Whereas in swing, a high level of activity may not be necessary to prevent trunk lean resulting only in further anterior pelvic tilt and hamstring lengthening. Indeed, Saunders, Rath and Hodges (2004) and Mann, Moran and Dougherty (1986) found the erector spinae is not active during the swing phase (a small portion of activity during forward swing likely corresponds to the contralateral foot strike), so if the erector spinae was weakened, it's possible the typical anterior pelvic tilt whilst running could still be maintained during the swing phase as it requires little force, whereas during stance where demands are higher (Saunders *et al.* 2005), trunk lean and the anterior pelvic tilt could increase if the erector

spinae was weakened. The simulation of Chumanov, Heiderscheit and Thelen (2007) does not consider the effects of greater erector spinae force during the stance phase of sprinting, where greater activity might reduce the trunk lean and thus hamstring lengthening.

Sherry and Best (2004) found anti trunk extension (i.e. isometric trunk flexion) and trunk rotation exercises combined with agility drills reduced hamstring reinjury rates compared to general strengthening and stretching of the hip and knee in the initial two weeks of returning to play (54.5% and 0% respectively) and one year after (7.7% and 70% respectively; $p < 0.05$ for both). Unfortunately, it's not clear whether strengthening of the trunk flexors and trunk rotators is responsible for the reduced injury re-occurrence rate because the agility programme would expose them to high-speed running, which appears to reduce injury risk (Duhig *et al.* 2016). Despite this, Chumanov, Heiderscheit and Thelen (2007) found increased oblique force (internal and external) can reduce BF lengthening during swing so it certainly seems a plausible outcome. For the iliopsoas, increased force during swing was found to increase hamstring lengthening (Chumanov, Heiderscheit and Thelen 2007), so weakness in this muscle would seem preventative rather than present a risk, at least during the swing phase, but as per the erector spinae effects, it may misleading to conclude this based on simulations limited to the swing phase only.

Schuermans, van Tiggelen and Witvrouw (2017) found prospectively injured participants initiate hip extension with the lumbar extensors ($p = 0.009$), which is the result of a delayed onset for hamstring activity rather than early lumbar extensor activity. This is highlighted by differences in onset times for each muscle. The effect size for onset time for the lumbar extensors is 0.11 (Cohen's *d*) larger in injured subjects but the for the hamstrings this increases to 0.72 (Cohen's *d*). Sole *et al*. (2011) observed a delay in hamstring onset time during transition from double to single leg stance but in participants with a HSI history, whereas Schuermans, van Tiggelen and Witvrouw's (2017) findings suggest the delayed hamstring onset is a cause of injury, though it's unclear why this would be. Perhaps the early lumbar activity, unopposed by the hamstrings, increases anterior pelvic tilt but this seems unlikely as

the difference is 12 ms and average EMG activity of the erector spinae was similar to the hamstring in the injured group (20 ± 10% and 19 ± 9% respectively) whereas in the control group erector spinae activity was slightly larger than the hamstrings (25% ± 30 and 22 ± 10% respectively), indicating greater anterior pelvic tilt would occur in the control group, not the injury group.

In summary, the evidence indicates muscles of the trunk could prevent hamstring injury and the mechanism may vary depending on the phase of the sprint and the muscles responsible. During stance, there is some empirical observations suggesting lower erector spinae activity, and thus force, may increase HSI risk by increasing forward lean and presumably hip flexion. During swing, the obliques or more generally the trunk flexor muscles, may act to prevent anterior rotation of the pelvis but the supporting empirical observations are confounded by an agility programme (Sherry and Best 2004) adding some doubt to the findings. Likewise, the empirical observations supporting erector spinae as a cause are far from definitive (Schuermans *et al.* 2017b; Bonte *et al*. 2015). There is some evidence that hip flexor weakness might prevent injury rather than cause it by reducing hip flexion during swing, but this needs further investigation to consider the potential effects that might occur during stance. To summarise, there is some limited evidence implicating the trunk flexors and extensors as potential causes of hamstring injury. Nonetheless, there is a clear need to consider more broadly the potential consequences of core muscle weakness in relation to anterior pelvic tilt, as per the conceptual framework of the HSI mechanism, across both the stance and swing phase of high-speed running.

2.4.2. The role of core muscles in pelvis kinematics

The thesis so far has referred to the core muscles without any specific definition to ascertain a broad understanding of the current literature. As the topic begins to narrow and direct its focus to the possible mechanisms of injury, it's important to define what will now be considered a core muscle.

2.4.2.1. Defining the core muscles

Considering the aforementioned research suggesting a possible mechanism involving changes to pelvis or trunk kinematics, the core is defined as muscles that attach to the ribs or thoracic and lumbar spine and attach to the pelvis. At the posterior, this includes the erector spinae muscles (ES), multifidus (MF) and quadratus lumborum (QL). At the anterior, this definition includes the internal and external obliques (IO and EO respectively), the rectus abdominis (RA) and the transversus abdominis (TrA). This definition aligns with the only prospective study to show an association between proximal muscles of the abdomen and back and HSI risk (Schuermans *et al.* 2017b), and Chumanov, Heiderscheit and Thelen (2007) who showed the theoretical potential for these proximal muscles to alter hamstring lengths through simulation. In addition to these muscles, the iliopsoas complex is included despite its inferior attachments below the pelvis. This was because the iliacus (IL) and psoas (PS) are considered a functional unit and when combined they have attachments to the lumbar spine (psoas) and the pelvis (iliacus) and meet the definition provided. Chumanov, Heiderscheit and Thelen (2007) also highlighted a potential role for more distal muscles such as the rectus femoris, adductor magnus and hip extensors to alter hamstring lengthening but their attachments are below the pelvis. Muscles with attachments below the pelvis can be trained with lower limb exercises, such as squats (Akaji *et al.* 2020), whereas this thesis was interested in the utility of core training, which tends to focus on actions of the trunk (Sato and Mokha 2009). For this reason, muscles with attachments below the pelvis are not considered core muscles for this thesis.

Table 4 and 5 describe the anatomical attachments of the anterior and posterior core muscles respectively and their functions in the sagittal plane. The sagittal plane was of interest as it captures

anterior rotation of the pelvis, along with lumbar spine and hip flexion, and knee extension. All of which have been proposed to have some involvement in HSIs. In brief, the abdominal muscles (RA, TrA, IO and EO) can flex the trunk and posteriorly tilt the pelvis, whereas the posterior muscles (i.e. ES, MF and QL) can extend the lumbar spine and anteriorly tilt the pelvis. The PS and IL are able to flex the hip and lumbar spine and increase anterior rotation of the pelvis. The following section will now detail the typical sagittal plane kinematics whilst running so that this can be related to activity in the core muscles and understand their precise role in sprinting.

Table 4: Anatomical details and actions of the anterior core muscles

Muscle	RA	TrA	EO	IO	PS	IL
Superior attachments (Medial for TrA)	Fifth, Sixth, and seventh costal cartilages	Linea alba Pubic crest via aponeurosis with IO	Ribs 5–12	Ribs 9-12 Ribs 7–9 via aponeurosis Iliopectineal arch	Anterior of transverse processes on L1-L5	Iliac fossa Iliac crest Sarcoilliac and iliolumbar ligaments
Inferior attachments (lateral for TrA)	Pubic crest	Seventh-Eleventh costal cartilages Thoracolumbar fascia Inner lip of anterior iliac crest Iliopectineal arch	Ribs 11–12 attach to anterior iliac crest Ribs 5–11 attach to anterior aponeurosis Pubic tubercle via aponeurosis	Thoracolumbar fascia Anterior iliac crest Pubic crest via aponeurosis with TrA	Lesser trochanter of femur	Lesser trochanter of femur
Sagittal Actions	Trunk flexion Pelvic posterior rotation	Increases intra-abdominal pressure Posterior pelvic tilt[a]	Trunk flexion Posterior pelvic rotation	Trunk flexion Posterior pelvic rotation	Hip flexion[b] lumbar flexion[b]	Hip flexion[c] Pelvic anterior rotation[c]

Note: [a]Franz *et al.* (2009); [b]Juker *et al*. (1998); [c]Andersson *et al*. (1995). Where a reference is not provided, the actions of a muscle are deduced from anatomical attachments.

Table 5: Anatomical details and actions of the posterior core muscles

Muscle	*Iliocostalis lumborum pars thoracis	*Iliocostalis lumborum pars lumborum	*Longissimus thoracis pars thoracis	*Longissimus thoracis pars lumborum	Multifidus	Quadratus lumborum
Superior attachments	Ribs 4–12	L1-L4 transverse processes and the thoracolumbar fascia lateral to vertebrae	T1-T5 transverse processes T6-T12 transverse processes and adjacent rib	Transverse and accessory processes of L1–L5	Spinous processes of vertebrae	Twelfth rib, T12 and transverse processes of L1-L4
Inferior attachments	Medial of iliac crest at dorsal segment	Medial of iliac crest at dorsal segment	T1–T6 attaches to L1–L5 T7–12 attaches to posterior surface of sacrum T12 also attaches to dorsal segment of iliac crest	L1–L4 attaches to medial of ilium next to sacrum via lumbar intermuscular aponeurosis L5 attaches to ventro-medial aspect of ilium and sarcoilliac ligament	Transverse processes 2–5 segments below superior attachmen and posterior surface of sacrum	Iliac crest level with L4
Sagittal Actions	Vertebrae extension Pelvic anterior rotation[a]	Vertebrae extension Pelvic anterior rotation[a]	Vertebrae extension Pelvic anterior rotation[a]	Vertebrae extension Pelvic anterior rotation[a]	Vertebrae extension Anterior pelvic rotation[a]	Vertebrae extension Pelvic anterior rotation

Note: *Collectively these muscles form the erector spinae; [a]Franz *et al.* (2009). Where a reference is not provided, the actions of a muscle are deduced from their anatomical attachments.

2.4.2.2. Normal lumbo-pelvic mechanics

Schache *et al.* (1999) reviewed hip, pelvis and trunk mechanics during running and later provided the first three-dimensional analysis of the lumbar spine in running (Schache *et al.* 2002). The kinematic findings of these two studies are combined in figure 4. The hip presents an uncomplicated pattern, extending throughout stance, peaking at toe-off, followed by flexion until ~80% of the gait cycle from which point the hip begins to extend again ready for touch–down.

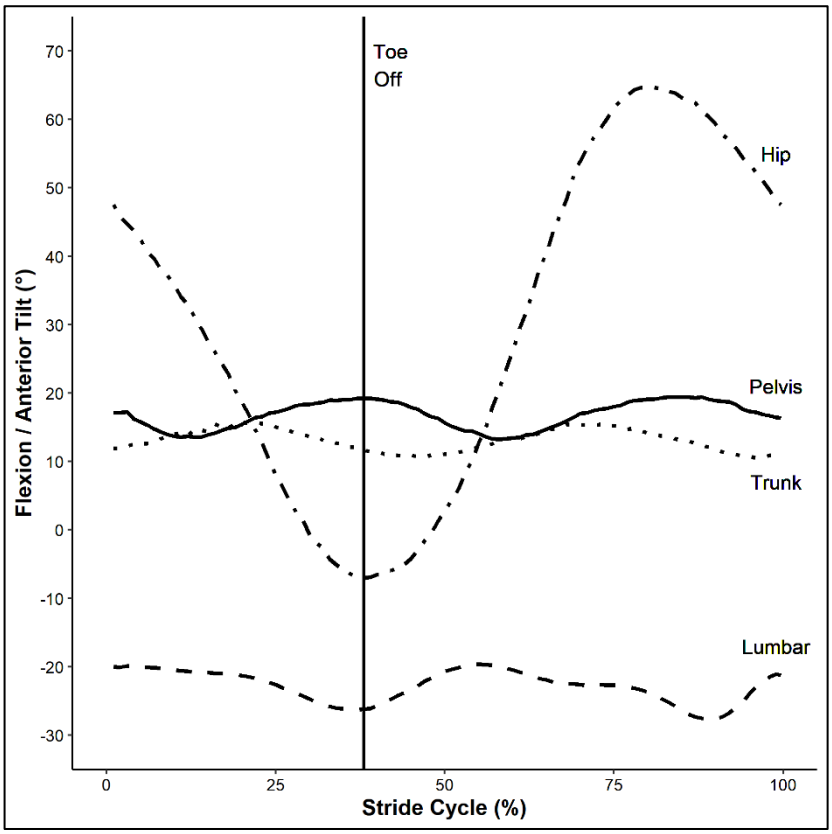

Figure 4: the kinematic waveform for the lumbo-pelvic-hip complex in the sagittal plane whilst running.

The pelvis is less straightforward. The pelvis begins to rotate posteriorly from touch-down to mid-stance, likely to facilitate closed chain hip extension. From mid-stance, the pelvis anteriorly rotates, peaking simultaneously with peak hip extension at toe-off. This pattern from posterior tilting to anterior tilting, is thought to arise as a mechanism of increasing stride length (i.e. distance covered during stance; Schache, Blanch and Murphy 2000). Indeed, peak hip extension is unchanged from walking to running (14.1° ± 6.4° to 14.6° ± 5.6°) but anterior pelvic tilt increases (6.8° ± 6.1° to 16.2°

± 6.5°), which allows for greater extension of the thigh (Franz *et al.* 2009). After toe-off, the pelvis returns to posteriorly tilting with hip flexion, but soon after anteriorly tilts to mid-swing where hip flexion peaks. It is suspected that the anterior rotation of the pelvis during swing reflects the contralateral hip extending in stance. At late swing, to the right of the curve, the pelvis begins to posteriorly rotate again with hip extension.

Changes in the lumbar spine angle can be difficult to distinguish from the pelvis, as Schache *et al.* (2002) calculated the lumbar spine angle relative to the pelvis angle. Thus, changes at the lumbar spine can result from rotation of the pelvis segment or rotation of the lumbar segment. The thigh is also calculated relative to the pelvis but it's much larger ROM makes this easier to distinguish. it's possible the changes in the lumbar angle waveform occur whilst the lumbar segment remains stationary in the laboratory reference frame because of changes in the pelvis. A cross-correlation using the pelvic data by Schache *et al.* (1999) and lumbar data by Schache *et al.* (2002) show a synchronous correlation (figure 5), suggesting that in normal kinematics, changes in lumbar motion occur simultaneously with changes at the pelvis (and vice-versa). If lumbar motion was independent of the pelvis, some lag between these two measures would be expected. In this respect, the trunk angle used by Schache *et al*. (1999) can provide a unique insight as it is relative to the laboratory reference frame—not the pelvis.

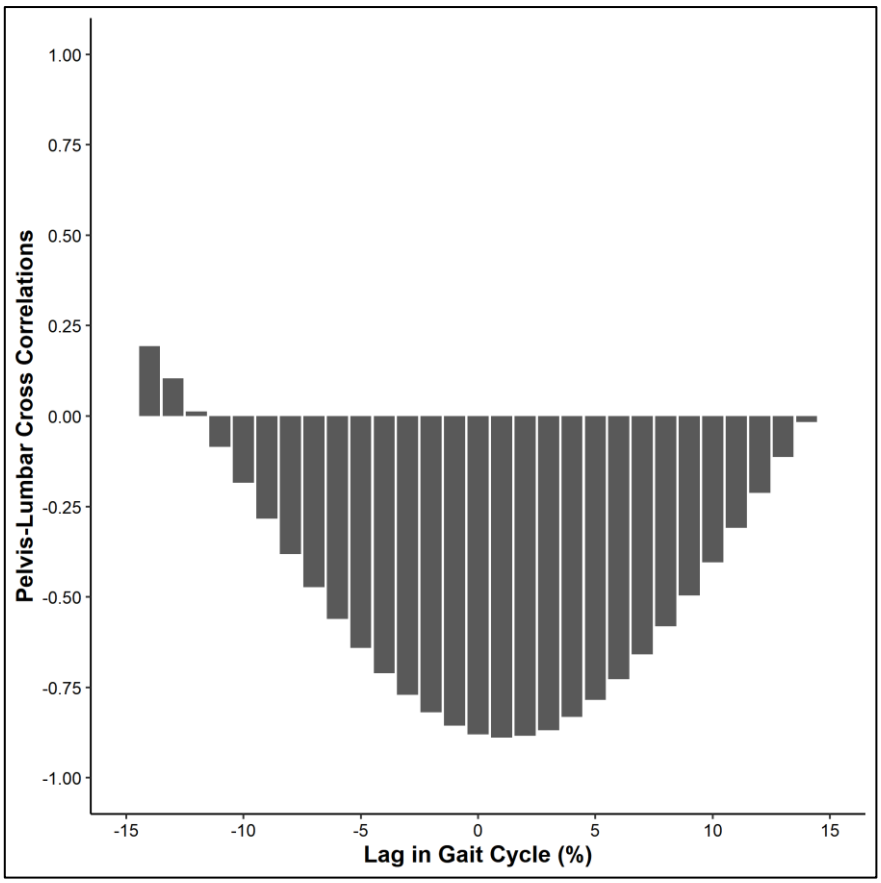

Figure 5: The cross correlation of pelvis-lumbar waveforms with a lag of ± 15% of the gait cycle.

From touch-down to mid-stance, the trunk leans forward whilst the pelvis posteriorly tilts (~3°) and the lumbar spine extends slightly relative to the pelvis (~1°). This would suggest the lumbar segment net extends ~4° in the laboratory reference frame whilst the trunk segment leans forwards. Indeed, the lumbar spine and trunk posteriorly accelerate at touch down (7.8 m/s^2; Kawabata *et al.* 2013), indicating the lumbar extension is to prevent the trunk from further lean, possibly to reposition the centre of mass (COM). Indeed, the posterior pelvic tilt with forward lean is thought to prevent large displacements in the COM so that changes in momentum are minimised (Preece, Mason and Bramah 2016a). In accordance with this notion, at mid-stance to toe-off, the trunk becomes more upright with further lumbar extension relative to the pelvis (~5°) and greater increases in anterior pelvic tilt (~3°). This suggests the lumbar segment net extends ~2°. Less than in early stance. Thus, despite the larger increases in relative lumbar extension at late stance, this is facilitated mostly by anterior rotation of the pelvis, which increases stride length at toe-off. On the other hand, during stance the lumbar segment must actively extend as the pelvis posteriorly tilts.

During swing, the trunk leans forwards with sharp rises in relative lumbar flexion, but this seems to be explained by increases in posterior pelvic rotation rather than flexion of the lumbar segment. Following mid-swing and after the trunk lean peaks, the trunk becomes more upright until touch-down and this does appear to occur with extension of the lumbar segment. A simple overview of the segments motion during running and sprinting can be found in table 6.

Table 6: Segment motion at each running phase

	Touch Down to Mid-stance	Mid-stance to Toe-off	Toe-off to Mid-swing	Mid-Swing to Touch down
Trunk	Leans	Extends	Leans	Extends
Lumbar	Extends	Extends	Flexes	Extends
Pelvis	Posteriorly	Anteriorly	Posteriorly	Anteriorly
Hip	Extends	Extends	Flexes	Extends

2.4.2.3. An analysis of core muscle activity and the associated sagittal plane kinematics

During the stance phase of running, ES and RA activity displays an anti-phase pattern. The ES is excited from touch down to mid-stance (Mann, Moran and Dougherty 1986) as the trunk leans forwards and the lumbar extends, and the RA is excited from mid-stance to early-swing (Mann, Moran and Dougherty 1986) as the trunk begins to become more upright. This suggests the ES and RA act to prevent excessive trunk motion, with the ES limiting trunk lean and the abdominals limiting trunk extension.

A more detailed analysis by Saunders *et al.* (2005) found abdominal excitation (RA, EO and IO) peaks at mid-stance, as the lumbar begins to extend, which likely coincides with trunk extension and anterior rotation of the pelvis (table 6). A peak in abdominal activity at this point, along with continued activation through to early swing, suggests it acts to prevent extension of the lumbar spine or anterior rotation of the pelvis, or both. Mann, Moran and Dougherty (1986) also speculated the RA prevents anterior pelvic rotation. It's unclear if this happens in reality as it would be counterproductive to performance, as anterior rotation allows for greater stride lengths (Franz *et al.* 2009). Instead, the purpose is more likely to decelerate trunk extension as the trunk becomes more upright.

Analysis from Saunders *et al.* (2005) show the MF and ES peak just prior to lumbar extension (relative to the pelvis) at approximately mid-stance. This almost certainly suggests the posterior core muscles act to prevent further trunk lean and the subsequent displacement in COM by extending the lumbar spine. In support of this, Thorstensson *et al.* (1982) found bilateral excitation of the lumbar extensors (MF and longissimus) increases with trunk lean whilst running**.** At the fastest speed (5 $m \cdot s^{-1}$) used by Saunders *et al.* (2005), ES and MF activity did not peak until after the trunk had extended, near toe-off. This shift in timing of the peak ES and MF excitation might occur to produce more anterior rotation of the pelvis at toe-off and increase stride length. Although previous studies have suggested little ES and MF excitation is needed to maximally anteriorly rotate the pelvis (19 ± 12 % and 24 ± 16 % of MVC respectively; Takaki *et al.* 2016) compared to the

excitation peaks as high as 90% of MVIC observed by Saunders *et al.* (2005). Though Sado (2016) suggested the lumbar extensors, which form part of the erector spinae, act not to produce anterior pelvic tilt but instead maintain it against the posterior pull of the hip extensors during stance. This may well require the high levels of activity observed by Saunders *et al.* (2005) at toe-off. Yet, findings have shown the hip is acting to flex rather than extend by mid-stance (Schache *et al.* 2011) suggesting the large erector spinae activity is unlikely to be in response to the hip extensor torque. A more likely reason is that the trunk lean becomes too great during stance at faster speeds and the ES must extend the spine for longer to become upright. Thus, the role of the ES and MF is that it acts to extend the lumbar spine and decelerates trunk lean during stance, a notion also put forwards by Saunders *et al.* (2005).

The patterns identified above appear to be mirrored during swing (Saunders *et al.* 2005), which might reflect the contralateral limb during stance as indicated by Mann, Moran and Dougherty (1986). Thus, the anterior and posterior core muscles appear active primarily in the stance phase rather than swing. This would certainly question the external validity of the increases in core muscle activity used by Chumanov, Heiderscheit and Thelen (2007) in their swing-phase simulation to ascertain HSI risk with core muscle force.

In previous sections where the mechanics of the hamstring were considered, the terminal swing phase of running was identified as possessing the greatest risk for HSI. Given the anterior and posterior core muscles are primarily active in stance (Mann, Moran and Dougherty 1986), it might seem they are unlikely to increase HSI risk. However, the early stance phase was also identified as a period of high risk (Ono *et al*. 2015; Orchard 2012) and the core muscles appear most active in this phase (Saunders *et al.* 2005). Furthermore, one should be careful not to assume the phases of the running stride with most risk during non-injuring runs as also having the greatest risk during injuring runs. It may well be that during injuring runs, the mechanics are altered such that the risk of injury occurs elsewhere in the stride cycle.

Whilst the abdominal muscles (RA, TrA, IO and EO) and posterior core muscles (ES and MF) appear to exclusively resist lumbo-pelvic motion in the sagittal plane, the IL and PS act to resist and produce movement. The IL and PS are excited prior to toe-off, when the hip is still extending (Andersson, Nilsson and Thorstensson 1997)**.** At this time, the pelvis is anteriorly tilting, and the iliacus could be partly responsible. Indeed, at the fastest speeds, where anterior tilt would be greatest to increase stride length, the iliacus excites before any other hip flexor (Andersson, Nilsson and Thorstensson 1997). However, many other hip flexors excite prior to hip flexion suggesting the muscle acts primarily to overcome hip extension and produce hip flexion during early swing (Andersson, Nilsson and Thorstensson 1997).

To summarise, the abdominal muscles (IO, EO, TrA, and RA) likely act to prevent excessive trunk extension from mid-stance to toe-off. These muscles have also been suggested to prevent anterior pelvic rotation at toe-off, but this seems counter-productive to sprint speed and unlikely. It seems more likely that that anterior pelvic tilt is actively sought, through the action of the iliacus, whilst it combines with the psoas to decelerate hip extension. The ES and multifidus appear to extend the lumbar spine from touch-down to mid-stance, in order to prevent excessive trunk lean and the subsequent displacement in the COM. Research has not yet investigated the QL during running but given its similar function and attachments to the ES and MF, it is expected to work in synergy with these muscles to extend the lumbar spine. Now the actions of core muscles during normal running have been considered, it is possible to consider the counterfactual where these muscles have insufficient force for their normal actions in order to propose the possible consequences of core muscle weakness for HSI risk during high-speed running.

2.4.3. consequences of core muscle weakness in the mechanism of injury

Now that the role of the core muscles has been made more apparent, the consequence of their weakness on HSI risk can be speculated on. The earlier section (2.4.2.3.) identified that the core muscles are excited most in the stance phase of running, and according to the causal model put forward in section 2.3.5., core muscle weakness is most likely to increase injury risk by increasing anterior pelvic tilt. With this in mind, the following section will consider how weakness in each of the core muscles could increase anterior pelvic tilt (if at all) during the stance phase of running. Postulated mechanisms will be evaluated against the empirical evidence already detailed in section 2.4.1. to evaluate its plausibility.

At first, the RA seems a likely candidate to increase anterior pelvic tilt if it were weak. The RA appears responsible to prevent excessive trunk extension (Saunders *et al.* 2005) but it can also produce posterior rotation of the pelvis according to its anatomical attachments (table 4). Presumably then, weakness would lead to an increase in anterior pelvic tilt. This idea has been put forward before (Mann, Moran and Dougherty 1986) but assumes the RA acts to resist anterior pelvic tilt in normal running, which seems unlikely given anterior pelvic rotation is thought to improve stride length (Franz *et al.* 2009) and thus performance. Of course, if the RA could limit anterior pelvic tilt between initial contact and mid-stance (when the thigh is relatively flexed and not in need of additional extension) then that may reduce the risk of HSI but EMG evidence suggests the RA is not excited during this time period (Mann, Moran and Dougherty 1986) and therefore not acting to prevent anterior pelvic tilt. In addition, abdominal strength, measured by the largest load it can resist to prevent anterior rotation of the pelvis, demonstrated no correlation to the pelvic tilt whilst running (r = 0.02–0.18; $p > 0.05$; Bickham, Young and Blanch 2000) suggesting participants with weaker abdominals do not have a more anteriorly rotated pelvis whilst running.

Even though RA weakness might not immediately result in further anterior pelvic tilt, one could be reasonably confident that lumbar extension would increase along with a more upright trunk just prior to toe-off. A more upright trunk has been shown to increase hip flexion torque just prior to

toe-off (~80% of stance; Leteneur *et al.* 2008) and the iliacus could induce greater anterior pelvic tilt (Andersson *et al.* 1995), but If this were the case one would still expect a correlation between trunk flexion strength and anterior pelvic tilt, which was not observed by Bickham, Young and Blanch (2000). Though the correlation was performed using baseline abdominal strength which might not be considered weak enough to observe changes in pelvic tilt. Nonetheless, although hip flexion torque was increased just prior to toe-off in participants with an upright trunk (Leteneur *et al.* 2008), no difference was observed at the moment of toe-off. Taken together, there is clearly an anatomical possibility for the RA to prevent anterior pelvic tilt, but the empirical evidence does not support this role. The external and internal obliques, along with the TrA, appear to share a common goal with the RA in the sagittal plane and act during mid-stance to toe-off to prevent excessive trunk extension (Saunders *et al*. 2005). Thus, weakness in these muscles is expected to have the same outcome as RA weakness in the sagittal plane. It may be easier to group these as the anterior core muscles in the following texts.

Similar to the anterior core muscles, the ES, MF, and QL share a similar function during gait and will be considered together as the posterior core muscles. Weakness in these muscles would appear to reduce anterior pelvic tilt according to their anatomical attachments (table 5) and lower the HSI risk. Indeed, at fast speeds it has been suggested these muscles might act to maintain anterior pelvic rotation and thus stride length (Saunders *et al.* 2005; Sado 2016). If these muscles were weak, then it seems the pelvis would posteriorly rotate from the hip extensor torque and lower HSI risk. However, the peak in ES activity whilst running has been suggested to be in response to the trunk forward lean during stance, in order to extend the spine (Saunders *et al.* 2005). As the ES activity peaks for this task, this is the action most susceptible to change due to weakness. Therefore, one can be confident that posterior core weakness would mean the lumbar segment would extend less during stance (i.e. be more flexed) and the trunk lean would increase.

Higashihara *et al.* (2015) found increased trunk lean (16.6° ± 1.7° to 33° ± 3.5°) is accompanied with increased anterior pelvic rotation (2.6° ± 6.7° to 12.8° ± 5.5°), and hip flexion (27.3° ± 7.5° to 42° ±

7.4°). This would appear to place the hamstrings at greater risk of injury. Higashihara *et al.* (2015) did not report the angle of the spine but it appears the spine was more flexed in the forward lean condition, as both trunk lean and pelvic tilt were measured about an axis in the laboratory reference frame. Therefore, if the trunk lean is greater than the pelvis angle, spine flexion must exist. In the forward lean condition, the trunk lean was greater than the pelvic tilt by 20.2° suggesting the spine was flexed by 20.2°, whereas in the upright condition the difference between trunk lean and pelvic tilt was 14°. Therefore, the spine appeared to be flexed by an additional 6.2° in the trunk lean condition. Further, the participants kinematics were captured soon after a block start position, where participants have been observed to have a flexed lumbar spine to achieve trunk lean (Debaere *et al.* 2013). Thus, this study suggests that increasing trunk lean with spine flexion is accompanied with further anterior pelvic tilt. This is somewhat supported by the empirical findings of Bonte *et al*. (2015) who found lower erector spinae activity and increased thorax flexion in hamstring injured subjects. Therefore, erector spinae weakness and a consequent increase in forward lean may well increase the risk of HSI during stance. The effects of lumbar extensor fatigue on running kinematics have previously been investigated (Hart et al. 2009) but the lumbar fatiguing protocol did not isolate the lumbar extensors and it's likely the hip extensors would have been fatigued, confounding the findings. Indeed, there is no correlation between isolated lumbar extension torque and trunk extension endurance (r = 0.06; p = 0.720; Conway *et al.* 2016).

Whilst kinematic data suggest anterior pelvic tilt occurs with greater spine flexion and trunk lean, it is useful to consider how this would ensue. A standing trial where the trunk was leaned forward (Δ 31.1°) resulting in spine flexion (22°) and anterior pelvic tilt (9°) found an increase in posterior leg muscle activity and a reduction in anterior leg muscle activity (Prior *et al.* 2014). Whilst the core muscles were not measured, it is suspected the anterior core muscles would lower their activation in order to facilitate lumbar extension, which might allow for further anterior pelvic tilt. This seems unlikely as it was suggested earlier that the RA may not act to prevent pelvic tilt, so the RA might not be active to a large extent to begin with. Somewhat paradoxically, erector spinae fatigue (a reduction in the maximal force generating capacity) could lead to an increase in force from these

muscles during the running stride that is greater than normally experienced, resulting in further anterior pelvic tilt. This scenario could occur if the rate of force development is impaired from fatigue such that lumbar extension and the deceleration of the trunk cannot occur as quickly, resulting in a more flexed lumbar spine and a greater forward lean. This could lead to greater forces than normal because of the increased gravitational moment acting on the trunk as suggested by Preece, Mason and Bramah (2016b). This idea is supported by observations during walking, where forward lean increases lumbar extensor torque at foot-strike and toe-off during walking (Leteneur *et al.* 2008) **al**though this would contradict observational findings that show injured subjects have lower erector spinae activity (Bonte *et al.* 2015). Though an increase in active force is not the only way lumbar extensor force might increase, as the longer ES lengths that occur with lumbar flexion would increase passive tension and also contribute to anterior pelvic tilt (McGill and Kippers 1994)**.** If the lumbar spine is fatigued to such an extent that the action becomes eccentric, this this too might increase forces to levels greater than normally experienced in running without lumbar extensor fatigue.

In addition, it's possible the pelvis becomes more anteriorly tilted with lumbar extensor fatigue not by producing anterior pelvic tilt but instead by reducing the ability to posteriorly tilt the pelvis. The increased trunk lean that consequences from lumbar flexion is expected to increase hip extensor demands (Preece, Mason and Bramah 2016b; Kluger *et al.* 2014; Leteneur *et al.* 2008) such that they are unable to produce the posterior pelvic tilt normally experienced. Lieberman *et al*. (2006) proposed the hip extensors act to limit trunk lean by posteriorly tilting the pelvis and Preece *et al.* (2016) suggested the posterior rotation of the pelvis at touch down limits anterior acceleration of the COM from trunk flexion. If ES weakness means the lumbar spine cannot contribute to decelerating the trunk lean, then the hip extensors will likely need longer to achieve the same deceleration. As time during the stance phase of running is limited by performance demands, this would mean there is less time to posteriorly tilt the pelvis. Further, the increased anterior displacement in COM with the greater forward lean is expected will increase the moment arm from the pelvis. Therefore, greater hip extensor force will be needed to posteriorly rotate the pelvis. The

result of these two factors would be a pelvis that undergoes less posterior rotation and thus appears more anteriorly tilted than in normal conditions. In support of this, participants with lower hip extension strength demonstrated increased lumbar spine flexion during vertical jump landings (8° ± 3° and 13° ± 3° for strong and weak groups respectively) and a reduced ability to decelerate lumbar flexion (peak flexion velocity was 143 ± 37 deg·s^{-1} and 221 ± 42 deg·s^{-1} for strong and weak groups respectively). There is some conflicting data that show a moderate correlation between hip extensor strength and trunk flexion whilst running (r = 0.55; Teng and Powers 2016), but this could suggest runners at moderate speeds with poor hip extension strength adopt a more upright trunk to compensate. Taken together, it seems fatigue of the erector spinae muscles would lead to an increase in trunk lean, which in turn contributes to a more anteriorly rotated pelvis. Though the precise mechanism is unknown, this may be by increasing erector spinae passive forces, reducing anterior core muscle activity, reducing the ability of the hip extensors to produce posterior pelvic tilt, or some combination of these factors.

According to the anatomy of the hip flexors, weakness is expected to reduce anterior pelvic tilt because the iliacus' superior attachment to the pelvis pulls it anteriorly (table 4). Whether this occurs in reality is unclear, as fatiguing the hip flexors did not change the mean pelvic tilt angle during walking despite reducing hip flexion velocity at pre-swing (169.5 ± 29.7 deg·s^{-1} to 133.2 ± 35.4 deg·s^{-1}; Akalan *et al.* 2016). Though the knee was more extended during swing which would lengthen the hamstrings and put them at risk of HSI (Akalan *et al.* 2016). Previous simulations have shown rectus femoris activity to increase to compensate for hip flexor fatigue (van der Krogt, Delp and Schwartz 2012), which might explain the increased knee extension during swing, but Akalan *et al.* (2016) observed no increase in knee extension torque after hip flexion fatigue. Thus, the increase in knee extension may have been an attempt to maintain stride length during walking as hip flexion is reduced. It is unlikely this would occur in running as foot strike ahead of the COM would result in large braking GRFs. Furthermore, the findings of Chumanov, Heiderscheit and Thelen (2011) suggest hip flexor weakness would reduce injury risk because the hip would be less flexed during swing.

Relying on the simulation of Chumanov, Heiderscheit and Thelen (2011) alone to infer consequences of core muscle weakness on HSI risk could be misleading as analysis was limited to the swing phase. For example, as the iliacus appears to prevent excessive hip extension, it's possible fatigue in these muscles will allow for further hip extension and in turn increase passive forces in the hip flexors. These passive forces could increase anterior pelvic tilt and the HSI risk but was not considered by Chumanov, Heiderscheit and Thelen (2011). Although hip extension flexibility is not correlated to anterior pelvic tilt during running (Schache, Blanch and Murphy 2000) and experimental evidence shows hip flexor stretching has no effect on anterior pelvic tilt whilst running despite improving hip extension flexibility by 10.7° ± 6.9° (Mettler, Shapiro and Pohl 2019).

This section has considered the potential effects of core muscle weakness on HSI risk. Trunk flexor weakness could allow for further anterior pelvic tilt due to their anatomical connections to the pelvis, but the empirical evidence and interpretation of the rectus abdominis activity suggests this is unlikely to occur, though empirical evidence has only considered baseline strength (Bickham, Young and Blanch 2000) rather than strength after fatigue where increased anterior pelvic tilt might begin to manifest. There is some evidence that erector spinae fatigue and the associated posterior core muscles would lead to reduced lumbar extension (more flexed lumbar spine) and trunk lean, which in turn could lead to an increase in anterior pelvic tilt. This notion is supported by some empirical observations (Bonte et al. 2015). Chumanov, Heiderscheit and Thelen's (2007) simulation suggested hip flexor fatigue would protect against HSIs during the swing phase as the hip would be less flexed, though weakness in these muscles also has the potential to increase anterior pelvic tilt during stance if hip extension is increased, which would generate passive forces pulling the pelvis anteriorly, but there is convincing evidence showing this is not the case. Considering the available evidence, posterior core muscle weakness (notably the ES) appears the most likely candidate to increase HSI risk but it should be acknowledged that anterior core muscle weakness might increase HSI risk too if the magnitude of fatigue in these muscles lowers strength values to less than what is observed at baseline in some participants (Bickham, Young and Blanch 2000). Unlike the anterior

and posterior core muscles, the hip flexors (iliacus and psoas) seem unlikely to increase HSI risk if weakened.

2.4.4. The plausibility of core muscle weakness during high-speed running

In the previous section (2.4.3.) it was shown that posterior core weakness is the most likely candidate between the core muscles to increase HSI risk, but trunk flexor weakness might be capable of increasing risk if fatigue lowers strength to values less than observed at baseline in some participants (Bickham, Young and Blanch 2000). Empirical findings suggested hip flexor weakness was unlikely to increase injury risk despite a possible mechanism existing (excessive hip extension). Regardless of the likelihood of a mechanism existing, it has no importance if the muscles are sufficiently strong such that the mechanism would never materialise. Therefore, the following section will consider the plausibility of core muscles being weak during high-speed running and sprinting, with the exception of the hip flexors (iliopsoas) as the evidence refutes the proposed mechanism for increased HSI risk with iliopsoas fatigue.

There are two ways which the core muscles may be considered weak. The most obvious of these would be when the core muscles maximum force production, commonly referred to as core strength, is insufficient for the tasks demands. The other possibility is when the core muscles become weak due to prolonged or repetitive actions, typically referred to as fatigue. The likelihood of the core muscles to be weak will be assessed on the evidence of their maximum strength relative to the demands during high-speed running and their potential to be fatigued.

The maximum strength of core muscles can be assessed by measuring the torque production in various movements (e.g. trunk flexion and extension), but whether the measured torque is sufficient requires further consideration. One way of assessing the sufficiency of a core muscles maximum force is by measuring their activation relative to a maximum action through EMG. Whilst EMG cannot tell you the force that is produced by a muscle (Vigotsky *et al.* 2018), muscles that need to produce more force increase excitation (i.e. rate coding or motor unit recruitment). If excitation

is normalised to a maximal voluntary contraction (MVC), then the capacity to produce force can be assessed. As excitation approaches 100%, less motor units can be excited to produce more force (assuming excitation-contraction coupling is intact) and thus their capacity to generate more force is smaller. Unfortunately, many studies do not normalise to a maximal reference contraction (Raabe and Chaudhari 2016; Andersson, Nilsson and Thorstensson 1997; Thorstensson *et al.* 1982). Cappellini (2006) showed trunk muscle EMG does increase with running speed but without an MVC to normalise to, comparisons between muscles are not possible. Behm, Cappa and Power (2009) did normalise to an MVC and found that the external obliques and erector spinae had similar activity on average during the running stride cycle (EO: 32.9% ± 22.9%; Upper ES: 40.4.% ± 28.0%; Lower ES: 36.2 ± 19.3) but average activity could be misleading as brief but high levels of excitation could be offset by generally lower levels of activity.

Fortunately, the work of Saunders *et al.* (2005) reported normalised peak core muscle excitation across multiple running speeds (table 7). The data showed the erector spinae, multifidus and external obliques display a high level of excitation when running at 5 $m \cdot s^{-1}$ suggesting little capacity for them to produce more force, which might become insufficient at greater speeds. This is concerning as soccer players reach speeds above 7 $m \cdot s^{-1}$ (Carling *et al.* 2016). The TrA showed values greater than maximum at 108% which suggests the MVC manoeuvre of a forceful exhale was not sufficient to generate a true MVC and is an inaccurate estimate of the muscles capacity to produce additional force (Saunders *et al.* 2005). The RA displayed notably low levels of activation suggesting it is the least likely to be weak (Saunders *et al.* 2005).

Table 7: Core muscle peak excitation relative to MVC during running

	Anterior muscles				Posterior muscles		
	RA	EO	IO	TrA	ES	Superficial MF	Deep MF
Peak activity (%)	31.4	97.8	53.0	108.0	87.7	95.8	75.2
SD (%)	19.3	31.7	69.3	76.7	146.3	64.0	102.7

Note: Data is taken from the fastest running trial at 5 $m \cdot s^{-1}$ from Saunders *et al.* (2005).

The work of Saunders *et al.* (2005) provides some insight into the capacity of each core muscle to produce sufficient force during high speed running but it is not possible to suggest how much additional force each muscle can produce until their capacity is reached, as the torque increase per a percentage point in activity is not equivalent across muscles due to differences in muscle physiology, such as the moment arm. Considering this and the precarious nature of EMG (Vigotsky *et al.* 2018), other research should be sought to support (or refute) these findings.

Sado *et al.* (2019) calculated the lumbosacral joint torques during high speed running in sprinters. It is possible to compare the mass normalised joint torques in sprinting to the maximum core muscle strength measured in soccer players by Fransson *et al.* (2018), and calculate if the sprinting torques are within their capacity (sprinting lumbo-sacral joint torque divided by maximum torque production). The lumbo-sacral joint torque estimates by Sado *et al.* (2019) were based on competitive sprinters where the speeds are expected to be higher than what soccer players experience, so the joint torques can be considered a worst-case scenario for soccer players. Lumbo-sacral extension torque plateaued above 6 $m \cdot s^{-1}$ and is estimated to be 61% of soccer players maximum force generating capacity. The lumbar-sacral flexor torque is estimated to be 44% of maximum force generating capacity at the fastest recorded speed (9.27 ± 0.36 $m \cdot s^{-1}$). Interestingly, lumbo-sacral rotation torque was highest and estimated to be 73% of the maximum force generating capacity and Sado *et al.* (2019) recommended strengthening of the rotator muscles, but not the trunk extensors or flexors. These results are aligned with the findings of Saunders *et al.* (2005), which showed high trunk rotator (external and internal obliques) and extensor (erector spinae and MF) activity but comparatively low trunk flexor (RA) activity. It appears the high oblique activity was not to flex the trunk but to rotate the thorax.

The estimates of core muscle force capacity during high-speed running may over-estimate their true capacity, as valid measures of spine flexion and extension require an immobile pelvis (Petersen, Amundsen and Schendel 1987). The device used by Fransson *et al.* (2018) to measure core strength did attempt to limit pelvis movement but used less restraints than in previously validated devices

(Petersen, Amundsen and Schendel 1987) and It is unclear whether this device isolates the spine entirely. Thus, soccer players true force generating capacity of the core muscles may be less than measured here.

Muscles that have larger demands are expected to hypertrophy as a physiological response. Similarly, those with little demands are not expected to hypertrophy. Therefore, changes in muscle CSA over a competitive season can be indicative of their demands. Over the course of a season in Australian football (which has similar demands to soccer) the CSA for internal oblique and erector spinae increased by 11.8% and 3.6% respectively (Hides and Stanton 2012), which is likely in response to the large demands imposed on them and reflects the aforementioned findings. Interestingly, the TrA reduced CSA by 21% which is in conflict with the 108% TrA activation whilst running observed by Saunders *et al.* (2005) and reinforces the idea that this EMG finding is an artefact of performing a submaximal MVC. The multifidus was also found to reduce CSA (4–11% depending on segmental level) despite high activity reported by Saunders *et al.* (2005), which might suggest the MVC for the MF was also inappropriate and the muscle has little demands during running. Whilst it is not possible to attribute the increases in CSA due to sprinting or running demands, they do align with the aforementioned evidence, and further, muscles that reduced CSA suggest that high-speed running (along with other movements too) do not place large demands on them, resulting in a loss of muscle size.

Overall, the evidence suggests the trunk rotators (internal and external obliques) and the trunk extensors (erector spinae) have the greatest demands during high-speed running and sprinting and the most likely to become weak. With the rectus abdominis, the principal trunk flexor, the least likely. Despite this, the evidence suggests all core muscles are sufficiently strong during high-speed running but that may not be the case during sprinting or running at very high speeds. The demands are also high enough that they might become weak from fatigue over the 90 minutes of soccer. In contrast to maximum strength, fatigue is a more complex and multifactorial phenomenon. Though for the purpose of this investigation, the precise cause may not be necessary as the magnitude of

force reduction is the primary concern (though knowledge of the underlying mechanisms of fatigue could be useful in the design of preventative interventions).

Fransson *et al.* (2018) investigated the reduction in lumbar/thoracic extensor and flexor torque after simulating the demands of soccer (Copenhagen soccer test) and found the trunk extensors reduced by 12 ± 3% and the trunk flexors by 10 ± 7% (standard deviations calculated from standard errors). This work is the first to measure core muscle fatigue after soccer and suggests the posterior core muscles are fatigued more than the anterior core muscles (Cohen's *d* of 4.00 and 1.43 respectively) and aligns with the high levels of activity in the posterior core muscles rather than the RA during non-fatigued running conditions (Saunders *et al.* 2005). This adds further evidence that the posterior core muscles are the more likely core muscle to be weak compared to the anterior core muscles. Although it is thought the obliques have similar if not greater demands to the trunk extensors (Saunders *et al.* 2005), this seems to be primarily for trunk rotation in the transverse plane (Sado *et al.* 2019), as the reduction in trunk flexion strength was less than the trunk extensors (Fransson *et al.* 2018).

Less than 0.2% of soccer match play is spent with the trunk extended (posterior to a vertical reference line), and considering the trunk flexors act largely to prevent excessive trunk extension, this is expected to place little demands on the trunk flexors over the 90 minutes (Oliva-Lozano *et al.* 2020). The remaining time is completed with the trunk leaned forwards, with 82% of game time spent between 20° and 40° of trunk lean (Oliva-Lozano *et al.* 2020). Therefore, it is logical the trunk extensors become more fatigued as it continuously acts to control the extent of trunk lean (Oliva-Lozano *et al.* 2020). Despite the trunk extensors fatiguing more than the trunk flexors, some soccer positions displayed a slightly more vertical trunk on average in the second half of soccer match play (~1.5°; Oliva-Lozano *et al.* 2020) rather than an increased trunk lean that would be expected if these muscles were fatigued (Winter, Gordon and Watt 2017). It may be that those positions perform less high-speed running (which would require forward lean) in the second half such that the average trunk angle is slightly reduced.

Further evidence exists to support the notion that the trunk extensors are fatigued more than the trunk flexors. Koblbauer *et al.* (2014) found peak trunk extension decreased when running to fatigue and peak trunk lean increased. Unexpectedly, trunk extension endurance was positively correlated to the change in trunk lean ($r = 0.74$; $p = 0.001$) but the increased hip extensor endurance (measured as part of the trunk endurance test) may have allowed participants to continue to run whilst simultaneously accumulating fatigue to the lumbar extensors. When hip extensor strength is measured, it has no correlation to trunk lean ($r = -0.22$; Ford *et al.* 2013).

Just as EMG can be used to calculate the peak demands on the core muscles, it can also be used to indicate the extent of fatigue. Saunders, Rath and Hodges (2004) reported the duration of core activity during running strides, with all core muscles active for over 70% of the gait cycle. Specifically, the RA, EO, IO and TrA were active for approximately 80-85% of gait whereas the ES was active for 70% and the superficial MF and deep MF active for 84% and 73% respectively. However, the duration of activity alone is misleading as it does not consider the magnitude of activity during this time. Saunders *et al*. (2005) later showed the RA had comparably smaller peak activity. Therefore, to ascertain the amount of physiological work performed by each muscle during running, it might be better to consider the integral of the normalised EMG activity with respect to time (% of gait). Unfortunately, there is no research reporting the integral of core muscle activity during running or sprinting but Behm, Cappa and Power (2009) reported average activity over a stride cycle, which can indicate the typical demands during running and thus fatigue. Behm, Cappa and Power (2009) reported the external obliques and erector spinae had similar activity on average during the running gait cycle (EO: 32.9% ± 22.9%; Upper ES: 40.4.% ± 28.0%; Lower ES: 36.2 ± 19.3) but no other muscle was measured.

Clearly, the erector spinae, or more broadly the posterior core muscles, along with the obliques are the more likely muscle to be of insufficient strength. Though weakness in the obliques appears primarily a concern for the thorax rotation rather than trunk flexion and the possible increased anterior pelvic tilt. Its possible synergistic muscles may be able to compensate for the ES preventing

any negative consequences of their weakness from materialising. Supporting this point, a simulation at slower speeds (2.80 ± 0.21 $m \cdot s^{-1}$) found the MF and QL increase their activity by ~40% each if the deep erector spinae fibres produced no force so that kinematics are maintained (Raabe and Chaudhari 2018). Unfortunately, the anterior muscles were not reported by the simulation so the ability to compensate for the anterior core muscles is not able to be compared, however, the low levels of RA activity in running suggest it is likely that oblique weakness could be more easily compensated for in comparison to erector spinae weakness, where MF activity in running is much higher (Saunders *et al*. 2005).

It was previously suggested that the trunk extensors and flexors both possessed a mechanism to increase HSI risk if they were to be weak but that the mechanism of injury from extensor weakness was more convincing, with some doubt that the mechanism with trunk flexor weakness would occur unless there was substantial fatigue in this muscle. After considering the potential for these muscles to be weak, the posterior core muscles (i.e. the trunk extensors) seem more likely to be weak based on their high level of activity during normal running and greater reductions in torque after soccer simulation compared to the trunk flexors. While trunk flexor fatigue was not substantially less compared to the trunk extensors (10 ± 7% compared to 12 ± 3%; Fransson *et al.* 2018), their low level of activity in normal running means the trunk extensors are the more likely muscle to become weak over the 90 minutes of soccer.

2.5. SUMMARY OF LITERATURE

Core strengthening is a globally adopted practice in soccer (Meurer, Silva and Baroni 2017; McCall, Dupont and Ekstrand 2016) often with the purpose of preventing hamstring injuries (Buckthorpe *et al.* 2019). It has even been referred to as 'the centrepiece' of training (Bliss and Teeple 2005). Yet, the evidence in support of core training to prevent HSIs is weak (Shield and Bourne 2018) and it is not known which core muscles should be strengthened despite some effort to identify them (Schuermans *et al.* 2017b). As a result, interventions often include a range of exercises. For example, The FIFA 11+ programme includes variations of the plank exercise with balance exercises without

any rationale regarding the target core muscle. Yet if a patient were prescribed a catalogue of treatments in hope it might prevent a disease it would be considered unethical.

The posterior core muscles and anterior core muscles, also known as the trunk extensors and flexors respectively, both seem capable of increasing HSI risk according to the model of hamstring strain injuries put forward earlier (figure 3) and their potential consequences if weak. Weakness in the posterior core muscles is expected to reduce lumbar extension and result in greater trunk lean, subsequently increasing anterior pelvic tilt. This notion is supported Bonte *et al.* (2015) who found lower ES activity and increased thorax lean in injured subjects, but is not a consistent finding as although Saunders *et al.* (2017b) found lower core muscle activity (1 time-dependent muscle activity vector) in prospectively injured subjects, they were unable to identify whether the obliques or ES was responsible. Anterior core muscle weakness could increase anterior pelvic tilt by producing less torque that posteriorly rotates the pelvis, though the evidence indicates this may not happen (Bickham, Young and Blanch 2000) and the low levels of activity in normal running suggest a greater magnitude of fatigue would be necessary before changes manifest compared to the posterior core muscles (Saunders *et al.* 2005). Furthermore, fatigue after soccer match play suggests the posterior core muscles are fatigued more than the anterior core muscles (Fransson *et al.* 2018). However, there is some doubt that the reduction in core muscle torque measured after soccer match play is valid, as the pelvis may not have been entirely immobilised during trunk extension measurements and could alter the conclusions of this review. Therefore, the aim of this thesis is to identify whether the anterior core muscles (trunk flexors) or posterior core muscles (trunk extensors) can increase HSI risk with the purpose of answering the overarching research question, “can core muscle weakness increase HSI risk in soccer players?”. The following individual research questions were addressed through empirical investigations:

1. Are the trunk flexors or lumbar extensors more likely to become weak from soccer match play?

2. Can the fatigue induced from soccer in the core muscle identified in study 1 be replicated in isolation using resistance machines for the purpose of causal inference?

3. Does the soccer equivalent magnitude of fatigue in the core muscle identified in study 1 increase anterior rotation of the pelvis whilst running?

4. Does the soccer equivalent magnitude of fatigue in the core muscle identified in study 1 affect hamstring torque production?

SECTION 3: EMPIRICAL STUDIES

3.1. THE EFFECTS OF SOCCER SIMULATION ON ISOLATED LUMBAR EXTENSION FORCE AND TRUNK FLEXOR FORCE

3.1.1. Introduction

Schuermans *et al.* (2017a) found increased anterior pelvic tilt whilst running in prospectively hamstring injured participants and it is suspected that a core muscle was responsible (Schuermans *et al*. 2017b). In the previous sections it was suggested that weakness in the trunk flexors and extensors due to fatigue over 90 minutes of soccer has the potential to anteriorly tilt the pelvis and increase the risk of HSIs. Between the trunk flexors and extensors, extensor weakness was identified as the most likely responsible for anterior pelvic rotation in high-speed running and sprinting. Though it would require a degree of fatigue to occur (Saunders *et al.* 2005). The increased anterior tilt observed by Schuermans *et al.* (2017a) was found during running without fatigue, but it may be that participants susceptible to injury represent a sub-set of the population which has less strength at rest, whereas for the average participant, weakness is likely to occur only after fatigue. It's possible the increased anterior pelvic tilt observed at rest may become even more pronounced after fatigue and at least partially explain the increase in injury incidence with match duration (Woods *et al.* 2004).

Fatigue in the trunk flexors and extensors has been demonstrated after soccer simulation (Fransson *et al.* 2018), but using measurements with little pelvic restraint, so the reduction in lumbar extensor torque could reflect reductions in hip extensor torque. Furthermore, the lumbar extensors (lumbar portion of erector spinae) are unique in that they are typically weak and cannot be strengthened without first preventing the sagittal rotation of the pelvis (Steele, Bruce-Low and Smith 2015; Fisher, Bruce-Low and Smith 2013) likely because of the dominance of the hip extensors as proposed by the deconditioning hypothesis (Steele, Bruce-Low and Smith, 2014). Whether fatigue in the lumbar extensors occurs in exercise without restraints might be a mundane notion to the unfamiliar reader, especially considering muscle activity is large during high-speed running (Saunders. *et al.* 2005) but it's worth remembering that despite inducing high activity, both Romanian deadlift training and hip

thrust training fail to increase lumbar extensor torque across the entire ROM (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013). Indeed, Vigotksy has shown that muscle activity from EMG cannot be used as an indicator of strength or hypertrophy outcomes (Vigotsky et al. 2018) and Steele, Bruce-Low and Smith (2015) highlighted a lack of agreement between exercises with high lumbar EMG activity and strength development.

The lumbar extensors are indeed a special case. Studies that claim to have fatigued the lumbar extensors or erector spinae have fatigued the hip extensors preventing causal inferences or used an arbitrary degree of fatigue that has little relevance for external applications (Hart *et al.* 2009; Champagne, Descarreaux and Lafond 2008). Although not certain, it seems likely lumbar extensor fatigue is possible as a study observed isolated lumbar extension (ILEX) fatigue without pelvic restraints after a series of kettlebell swings (Edinborough, Fisher and Steele 2016), but no research has examined the fatiguing potential of exercise outside of the resistance training modality, such as soccer. In addition, the magnitude of fatigue in the lumbar extensors in comparison to the trunk flexors is unclear and could have implications as to whether the lumbar extensors are even likely to become weak after soccer.

It is anticipated that the lumbar extensors become fatigued as they repeatedly extend the spine to control the forward lean during stance, whereas the trunk flexors likely act to limit trunk extension from mid-stance to toe-off (as suggested in section 2.4.2.3.). Theoretically, repeated running and sprinting over a 90-minute duration is a potent prescription for inducing lumbar extensor and trunk flexor fatigue. However, the demands of soccer are variable and not conductive to systematic investigation for causal effects. The coefficient of variation in sprints between match play is large (37.1%; Carling *et al.* 2016). An alternative that would provide proof of concept with an estimate that, on average and over many games, should theoretically be accurate is soccer simulation. Such protocols are used extensively in the literature for investigating similar variables (Silva *et al.* 2018; Fransson *et al.* 2018). Soccer simulations allow an experimenter to standardise the dose of activity across individuals and infer precise estimates of their effect. Therefore, the aim of this study is

twofold. First, to compare maximum ILEX torque between soccer players and non-soccer players to see if soccer participation and the involved high-speed running has not conditioned these muscles. Second, to assess whether lumbar extensor fatigue is possible after performing a soccer simulation and if so, comparing the magnitude of fatigue to that in the trunk flexors. It is hypothesised that soccer is not sufficient to strengthen the lumbar extensors as the high activity in strength training exercises fail to strengthen these muscles. Also, it is hypothesised that fatigue will occur in the lumbar extensors and the trunk flexors, but the magnitude of fatigue will be greater in the lumbar extensors.

3.1.2. Method

3.1.2.1. Study design

The investigation took the form of two independent studies. The first was measured isolated lumbar extensor torque before and after a soccer simulation in a repeated measures design, along with heart rate for comparison of the internal physiological demands. The second study implemented a repeated measures design to measure trunk flexion endurance before and after soccer simulation, as an indicator of fatigue in these muscles. Maximum hand grip strength was also measured before and after soccer simulation as a measure of non-local fatigue. This allowed for tests to see whether fatigue in the lumbar extensors and trunk flexors force were due to non-local factors.

3.1.2.2. Study 1: Participants

Twelve, male amateur soccer players (age: 20 ± 4 years; Stature: 179.4 ± 5 cm; mass: 72 ± 7 kg) were recruited for this study (five defenders; three attackers; four midfielders). Participants were obtained by contacting local soccer teams or through direct contact and word of mouth. Inclusion criteria required participants to be currently assigned to a club and were excluded if they had any lower limb or back injuries. According to the English national league system, most players most participated at level 10 (mode) and ranged between levels 8–15. Three participants competed in university or college leagues.

3.1.2.3. Study 1: Procedure

This study took a within-subjects repeated measures design. ILEX strength was measured pre- and post a soccer simulation protocol (Soccer-specific Aerobic Field Test [SAFT90]) and heart rate was measured throughout. After an initial briefing, participants completed a physical activity readiness questionnaire and were judged against the participation criteria. Participants were asked to attend the university laboratory on two occasions separated by at least 72 hours. The initial session familiarised participants with the procedure for ILEX testing and the soccer simulation to ensure reliability. The device (MedX, Ocala, FL, USA) isolates movement to occur in the lumbar spine only through a series of restraints that prevents pelvic rotation (figure 6) and has previously demonstrated strong test-retest reliability (r = 0.81– 097; Graves *et al.* 1990). Likewise, our laboratory has reported similar reliability (within-day r = 0.9 –0.99). The device has a minimum detectable change of 1286 N·m·deg according to the calculations of Weir (2005) and the raw control group test-re-test data of previous work (Stuart *et al.* 2018; Edinborough, Fisher and Steele 2016). Heart rate (TS1, Polar, Finland) was recorded every 15 minutes throughout the SAFT90 including the half-time 15-minute rest period. Upon completion of the SAFT90, participants were allocated

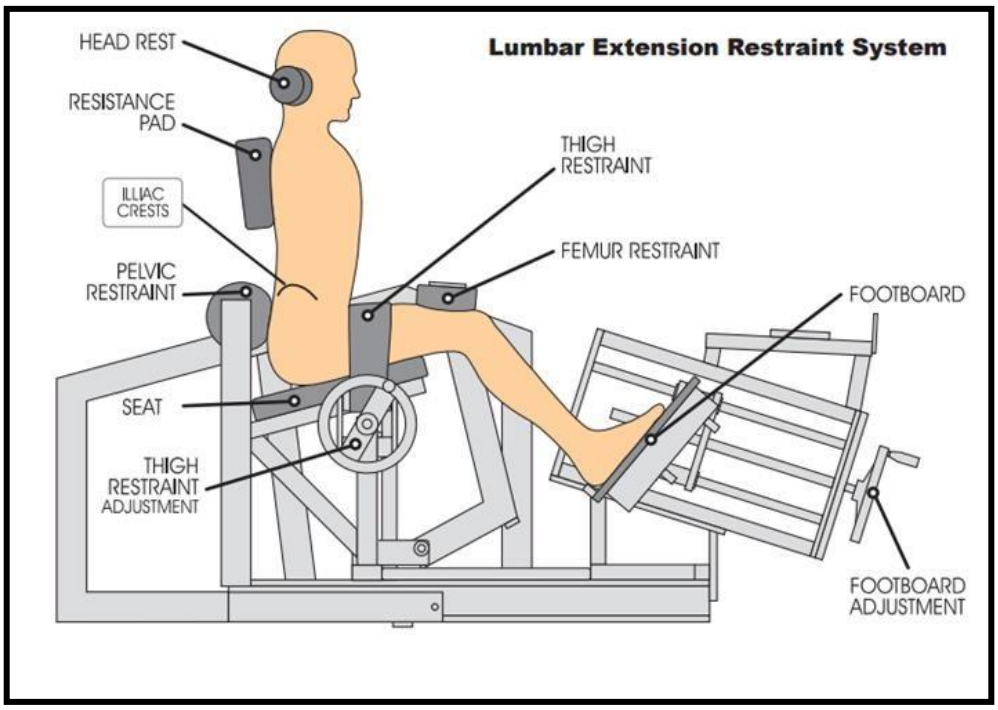

Figure 6: A schematic representation of the ILEX device and its restraints

five minutes rest before repeating the ILEX isometric strength test as per baseline. The five minutes rest allowed sufficient time for the participant to be re-positioned and secured in the ILEX device.

3.1.2.4. Study 1: Lumbar extension strength testing

The testing procedure for ILEX strength consisted of a dynamic warm up of eight repetitions across a full ROM using a load of 27 kg at a repetition duration of 2:4 s for concentric and eccentric portions respectively. Immediately following the warmup, lumbar extension strength was measured isometrically across the full ROM (typically 72°–0°), beginning in full flexion and progressively working towards full extension at 12° intervals. At each angle participants were asked to gradually increase the force up to maximal effort over a three second duration and verbal encouragement was provided throughout. Between each angle participants rested for approximately 10 seconds, during which they were passively moved across the ROM. In the following session, baseline ILEX strength was recorded by repeating the procedure in the familiarisation trial.

3.1.2.5. Study 1: Soccer simulation

After baseline testing, participants were allocated a brief seated rest period and were fitted with a heart rate monitor. At the end of the brief rest period baseline heart rate was measured and the soccer simulation protocol began. The soccer simulation (SAFT90; Lovell, Knapper and Small 2008) is a 90 min protocol separated by a 15-minute passive rest after 45 minutes (details of the course are provided in figure 7). The 15-minute passive rest was to represent the half-time interval in soccer. The Loughborough intermittent shuttle test (LIST) and the SAFT90 are the principle simulations used by prior research (85% of all on-field simulations; Silva *et al.* 2018), but unlike the LIST, the SAFT90 has an activity profile more specific to soccer, with a duration of 90-minutes (plus 15 minutes rest) and change of direction tasks compared to the LIST's linear movement profile and frequent rest periods.

The SAFT90 consists of repeated linear movements and alternating movements (side-steps, change of directions, and backwards running) with commands provided via pre-recorded audio. A variety

of speeds were required consisting of stationary, walking, jogging, striding, and sprinting. Participants were asked to maintain the required effort: if they could not keep up with the speed of the instructions and would catch up on slower phases such as walking, although this was limited to the faster 'stride' (90-95% effort) and 'sprint' (100% effort) portions of the simulation. This procedure has been used extensively in the literature to simulate football and allows for comparable results between research (Rhodes, McNaughton and Greig 2019; Lovell *et al.* 2018; Marshall *et al.* 2014; Small *et al.* 2009) and has been shown to be valid with regards to the physiological and mechanical demands (Lovell, Knapper and Small 2008).

The simulation was completed indoors on hard flooring to remove variability in the surface conditions due to weather. For the protocol to fit in the laboratory space, the protocol was modified in a similar method to (Azidin *et al.* 2015). The length of the course was reduced by 5 m, from 20 m to 15 m. To ensure the total distance covered remained similar, participants were required to travel out the 2 m cone and return to the start before beginning the next instruction. Total course length was reduced by 6 m but added an additional three changes of direction for every completion of the course. This was not considered to affect the validity of the simulation given match-to-match variability is large in soccer (Carling *et al.* 2016).

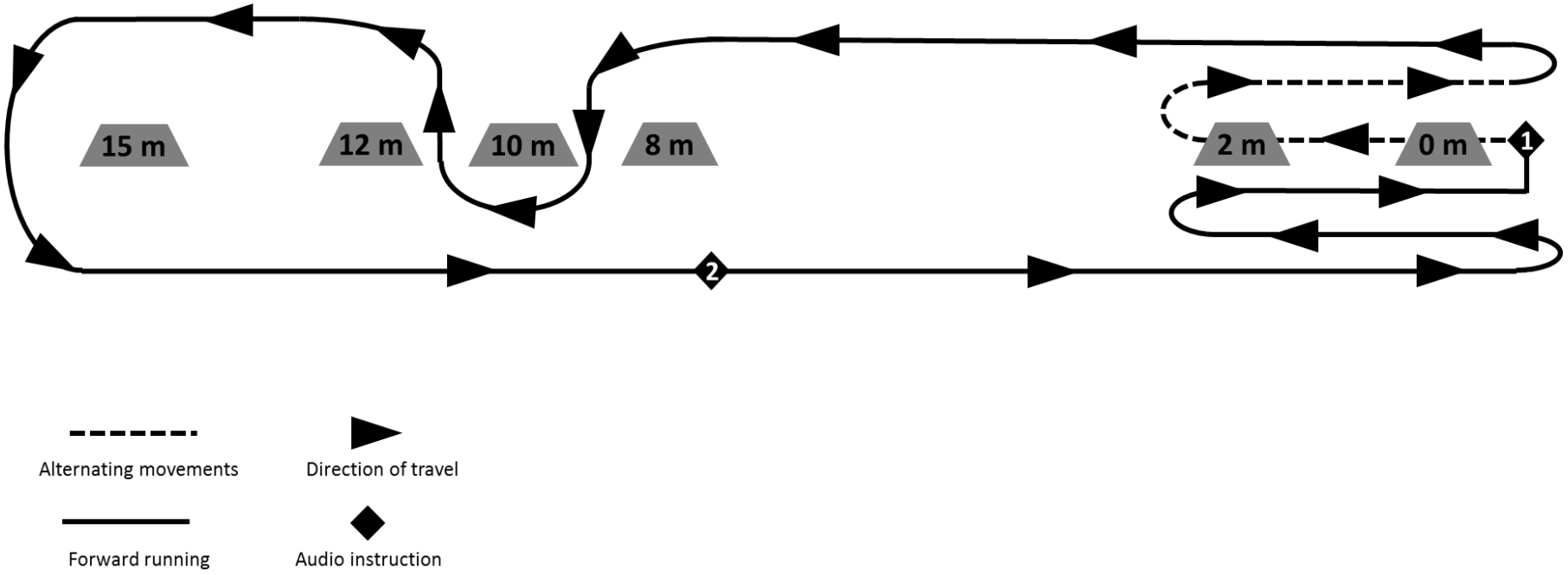

Figure 7: The adapted SAFT90 protocol

3.1.2.6. Study 1: Data analysis

A strength curve for the participants' ROM was generated using the peak torque at each angle. ILEX strength was measured as the area under the curve using the trapezoidal method and the outcome was labelled as the strength index (SI). The area for a single trapezoid (A) is calculated as:

$$A = \frac{a+b}{2}h \tag{3}$$

Where *a* and *b* are the peak torque values for two consecutive angles and *h* is the difference in degrees between the two angles, which is constant at 12° in this scenario. The SI was equal to the sum of these trapezoidal areas.

3.1.2.7. Study 2: Participants

Ten, male amateur soccer players (age: 22 ± 4 years) were recruited for this study (three defenders; four attackers; three midfielders). Participants were obtained by contacting local soccer teams or through direct contact and word of mouth. Inclusion criteria required participants to be currently assigned to a club and compete as an outfield player, and were excluded if they had any lower limb or back injuries. According to the English football league system, most players most participated at level 10 (mode) and ranged between levels 7—15.

3.1.2.8. Study 2: Procedure

Trunk flexion endurance and maximum hand grip strength was measured pre- and post the SAFT90 soccer simulation. After an initial briefing, participants completed a PAR-Q and were judged against the participant criteria. Participants were initially familiarised with SAFT90 protocol (if they were not previously) and the hand grip and trunk flexion endurance tests.

3.1.2.9. Study 2: Trunk flexion endurance and hand grip strength testing

Trunk flexion endurance was measured using McGill's trunk flexion endurance test, which has shown to have good reliability (ICC: 0.97; McGill, Stuart M., Childs and Liebenson 1999). During pilot studies, a reverse Biering-Sørensen test was trialled but participants reported discomfort in the

lumbar spine as the cause of test cessation rather than fatigue. When trialling McGill's isometric trunk flexor hold, participants reported that it primarily fatigued the hip flexors. Therefore, Mc Gills' isometric trunk flexor task was adapted to limit contributions of the hip flexors and control the change in lumbar lordosis angle. Participants laid supine with their palms pronated and shoulders relaxed. Participants were instructed to 'curl-up', raising their scapulae off the ground and allowing their fingers to slide forwards as the trunk was displaced. The change in lordosis angle was standardised by ensuring participants' third finger displaced by 10 cm, confirmed by contacting a wooden block measuring 10 cm away from the participants' third finger when laying silently in supine. A 10 cm displacement was chosen as it increases activation of the lower abdominal muscles, which attach to the pelvis, compared to 5 cm or 15 cm distances (Parfrey *et al.* 2008). To minimise hip flexor involvement, knees were bent to approximately 90° and feet had to remain in contact with the ground without external fixation (i.e. no hip flexion was allowed; Parfrey *et al.* 2008). Participants also had a 5 kg weight centred on their sternum to add additional load. In pilot testing, some subjects could maintain the task for long periods of time due to familiarity with it in their own training. The addition of the weight increased the demands and prevented task failure due to factors other than fatigue (e.g. tedium). Once a 10 cm displacement had been achieved with the third finger, the time was recorded until the participant could no longer maintain contact with the wooden block. Throughout the task, participants were given strict instructions to relax their shoulders and elbows and this was monitored by the investigator throughout. Participants were familiarised with the task prior to completing the test and verbal encouragement was provided throughout. All participants received a demonstration of the task and practiced with feedback the until the correct technique was used.

Hand grip strength was measured in the dominant hand using a dynamometer (5101 Grip-D, Takei, Japan) with the shoulder adducted and the elbow flexed to 90°. Participants were encouraged to provide a maximal effort over 3 s. Participants were given one practice attempt, followed by three recorded attempts. A rest period between each attempt was allocated.

After measuring hand grip and trunk flexion endurance, participants completed the SAFT90 protocol identically to that in study 1 of this investigation, including the 15-min rest to represent half time. Upon completion of the SAFT90, participants immediately repeated the three measures of hand grip strength. After five minutes since completing the SAFT90, participants repeated the trunk flexion endurance task. The 5-minute delay was chosen to align with the 5-minute delay used when measuring ILEX fatigue in study 1 and allowed for a comparison between the two measures. Hand grip strength was averaged across the three attempts using a mean in each condition and this value was used for statistical analysis.

3.1.2.10. Statistical analysis

For study 1, statistical analyses were performed using SPSS (Version 25; IBM Corp. 2019). To assess whether soccer players had similar lumbar extensor force to others, the baseline ILEX SI was compared to the ILEX SI in other populations, measured in previous investigations. These were asymptomatic individuals from Conway *et al.* (2018), and recreationally strength trained, non-competitive powerlifters and competitive powerlifters from Androulakis-Korakakis *et al.* (2018). The assumption of normality for all baseline ILEX SI data was assessed through a Shapiro-Wilk's test, which failed to reject the assumption of a normal distribution for all but the asymptomatic group (W = 0.935, df = 42, p = 0.019). Therefore, baseline SI data were reported as means and SDs apart from the asymptomatic group where data was represented as medians and IQRs. To test the hypothesis that soccer players ILEX SI differs to asymptomatic controls, a two-sided Mann Whitney U test was used. If this was significant then comparisons were made between soccer players and recreationally trained subjects using a two-sided independent t-test. Both inferential statistics had an alpha of 0.05.

For the change in ILEX SI after soccer simulation, the assumption of normality was tested through a Shapiro-Wilks test, which failed to reject the assumption of a normal distribution (W = 0.915; df =10; p = 0.280) and was confirmed through visual inspection. Therefore, descriptive statistics for the change in SI scores are presented as mean and standard deviations. SI change scores were

assessed using a single sample t-test against a value of 0 and deemed significant if the probability of these findings under the null hypothesis (no change) were less than 5% ($p < 0.05$). The precision of the findings was assessed through a 95% confidence interval. Heart rate data were reported descriptively to assess the external validity of the study by comparing to semi-professional soccer players completing the SAFT90 (Lovell, Knapper and Small 2008).

For study 2, statistical analyses were conducted in JASP (Version 0.14.1). A Shapiro-Wilk's test was unable to reject the assumption of normality for the change in hand grip data (W = 0.959; $p = 0.776$) but did reject a normal distribution for the change in trunk flexion data (W = 0.767; $p = 0.006$). A one-sided one sample t-test was used to test the null hypothesis that the change in hand grip strength was greater than 0 (i.e. hand grip strength improved). A one-sided Wilcoxon signed-rank test was used to test the null hypothesis that the change in trunk flexion endurance time was greater than 0 (i.e. trunk flexion endurance improved). Both tests had an alpha of 0.05.

The change in trunk flexion endurance time, change in ILEX SI, and change in hand grip strength after soccer simulation were converted to percentage change to allow comparisons between each muscle. Percentage change data for each muscle was normally distributed according to Shapiro-Wilk's test ($p > 0.05$) but trunk flexion change had a positive skew (1.27) on further inspection. If trunk flexion endurance or ILEX SI were found to decrease after the SAFT90, then a one-sided independent t-test was used to test the null hypothesis that the percentage reduction in either ILEX SI is less than the percentage reduction in non-local muscles (change in hand grip strength), or a one-sided Willcoxon signed rank test would be used to compare trunk flexion data to non-local muscles. This was a more rigorous test of whether local lumbar extensor or trunk flexor fatigue occurred after the SAFT90 or whether non-local mechanisms were responsible. Descriptive statistics for the percentage change for all muscles was reported using medians and interquartile ranges for comparison.

3.1.4. Results

Descriptive statistics for baseline ILEX SI at baseline for soccer players compared to other populations are presented in table 8. Inferential statics rejected the null hypothesis that soccer players SI does not differ to asymptomatic populations (asymptotic p = 0.02) but was unable to reject the null hypothesis that soccer players SI do not differ to recreationally trained subjects ($t_{(45)}$ = −1.127; p = 0.266). A visual comparison of SI values between populations (including powerlifters) is presented in figure 8.

Table 8: Comparison of Isolated lumbar extension SI between different populations

Sample	Mean SI (N·m·deg)	SD	N	Age (SD)
Soccer players	21836	5509	11	20 (4)
Asymptomatic*	13925	10519	42	30 (12)
Recreationally trained	23771	4823	36	25 (6)
Non-competitive powerlifters	23019	6843	10	24 (3)
Competitive powerlifters	22815	5812	13	32 (8)

**Note: ***SI data for asymptomatic sample is presented as Median and IQR as data was not normally distributed.

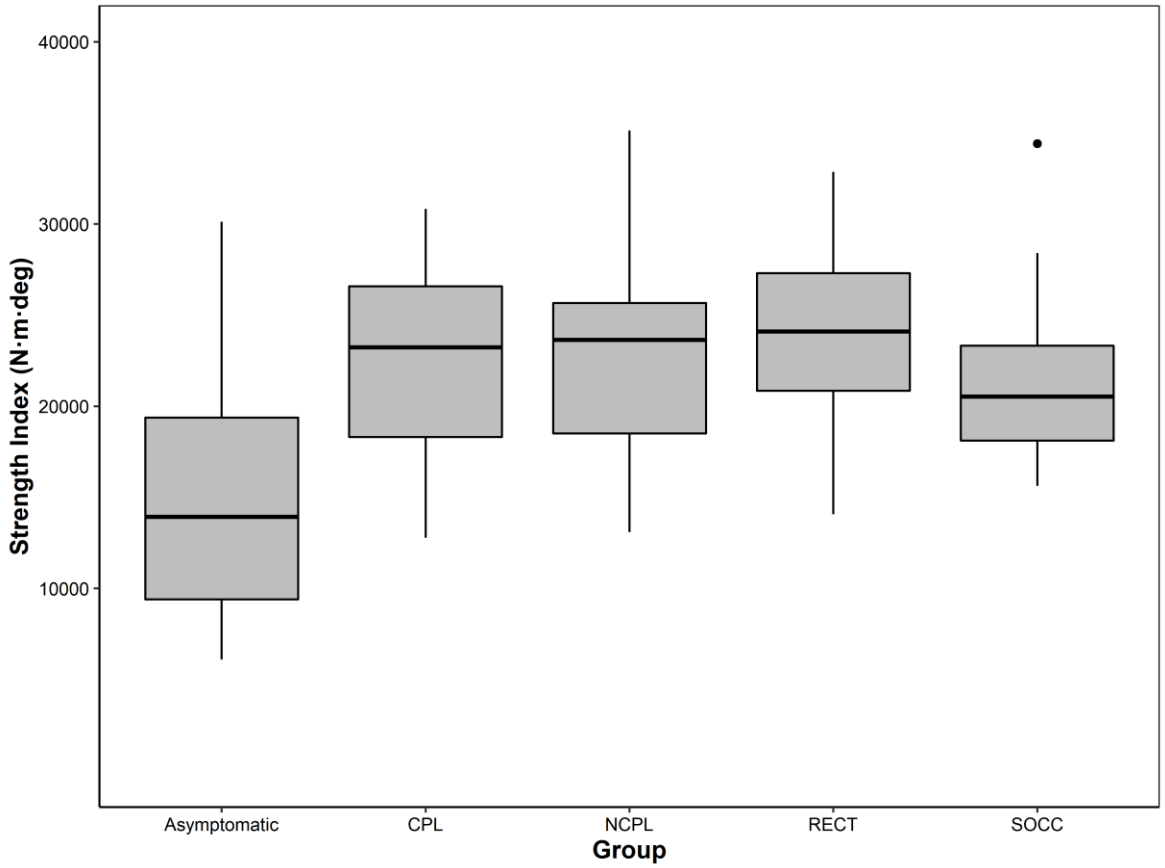

Figure 8: Isolated lumbar extensor SI between populations. CPL: competitive powerlifters; NCPL: non-competitive powerlifters; RECT: recreationally strength trained; SOCC: soccer players

In the measures of ILEX SI after soccer simulation, one outlier was removed due to an increase in force after fatigue that exceeded the typical minimum detectable change (figure 9), indicative of a true improvement. During further testing it was confirmed that the initial test was not reflective of their maximal strength. Therefore 11 participants were analysed for changes in SI. All subjects achieved a full ROM on the ILEX device. With the outlier removed, the mean change in SI scores after soccer simulation was −2767 ± 1860 N·m·deg, or 13% of baseline, and was statistically significant ($t_{(10)}$= −4.933; p = 0.01). According to the 95% CI (−1517 to −4017 N·m·deg), no more than 5% of studies will experience a mean reduction in strength less than 1517 N·m·deg if this study were to be repeated indefinitely using the same method and sample. Heart rate averaged 166 ± 3 b·m^{-1} across all time points which is descriptively similar to the 162 ± 2 b·min^{-1} reported in semi-professional soccer players performing the SAFT90 protocol.

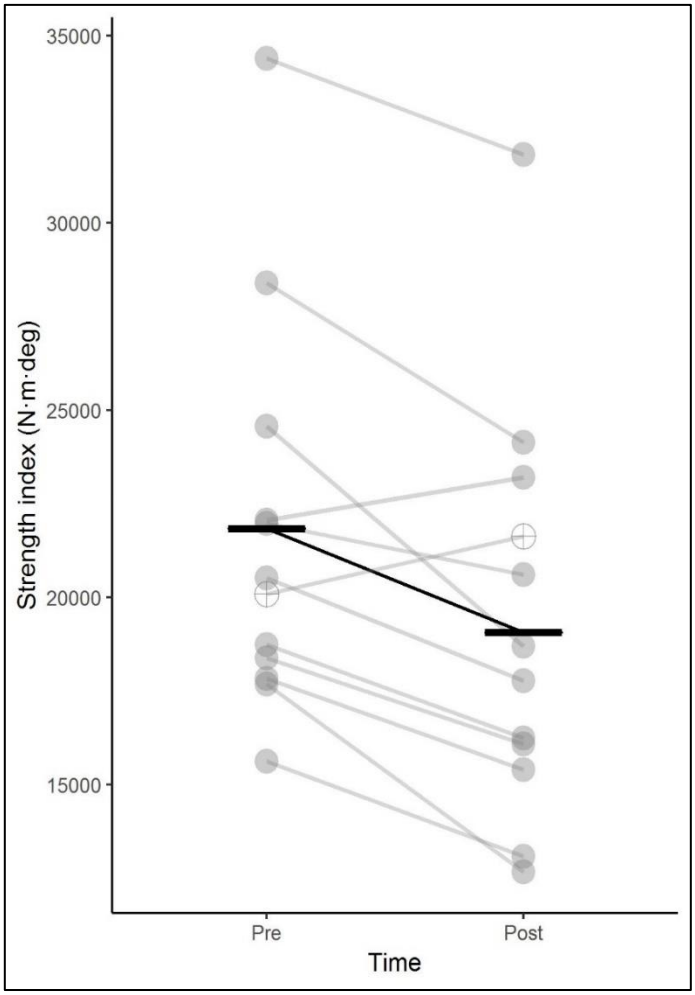

Figure 9: Individual and mean response in SI after the SAFT90 protocol. **Note:** Bold line represents mean outcome. Open circles represent the excluded outcome.

The median trunk flexion endurance time at baseline was 63 s with an interquartile range of 46–122 s. The median change in trunk flexion endurance time was −3 s with an interquartile range of −8–7 s. Inferential statistics for the change in trunk flexion endurance time failed to reject the null hypothesis that the change in time was greater than 0 *(V* = 25; p = 0.419).

The mean hand grip strength at baseline was 41.8 ± 5.6 kg. The mean change in hand grip strength was -2.2 ± 4.1 kg. Inferential statistics for the change in hand grip also failed to reject the null hypothesis that the change in hand grip strength was greater than 0 ($t_{(9)}$ = −1.664; p = 0.065).

A one-sided independent sample t-test rejected the null hypothesis that the percentage change in handgrip strength is less than the percentage change in ILEX SI (i.e. hand grip strength reduced more; $t_{(19)}$ = 2.072; p = 0.026). Comparisons between the change in trunk flexion time and hand grip strength were not conducted as trunk flexion endurance was not found to statistically reduce after the SAFT90. Comparisons of the percentage change in trunk flexion endurance time, change in ILEX SI, and change in hand grip strength are presented visually in figure 10. The median change in the trunk flexion endurance time was similar to the change in hand grip strength (−4.5% [29%] and −6%

[12.8%] respectively). The change in ILEX strength was comparatively much greater at −13% (5.5%) with only one participant displaying an increase in ILEX SI after the SAFT90.

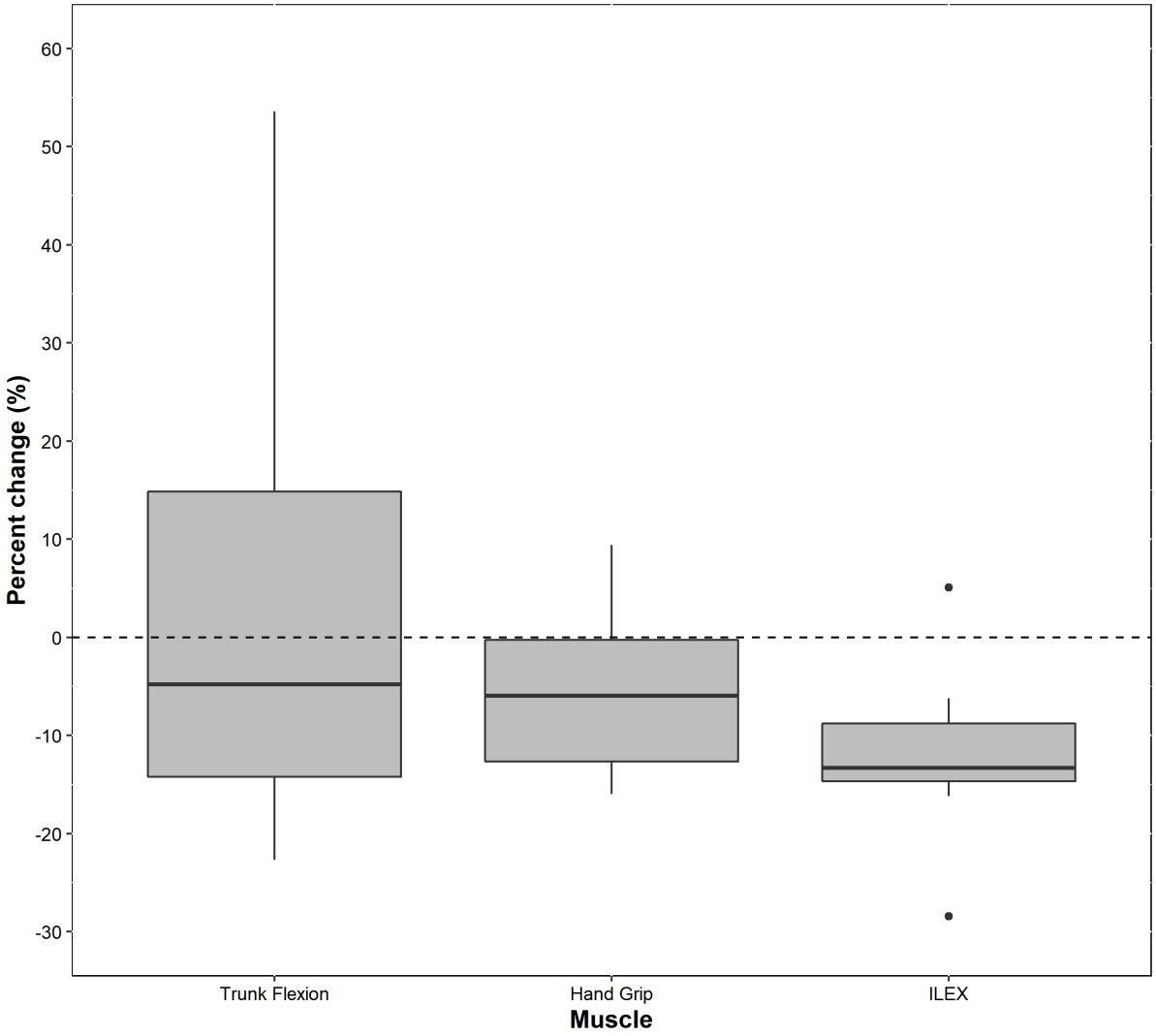

Figure 10: Percentage change for each muscle after the SAFT90. Dashed horizontal line intercepts at 0 as a reference of no change. ILEX: Isolated lumbar extension.

3.1.5. Discussion

This study had multiple aims. The first was to compare maximum isolated lumbar extensor torque between soccer players and non-soccer players. Inferential statistics revealed isolated lumbar extensor torque is greater in soccer players compared to asymptomatic controls from Conway *et al.* (2018) but is not different to recreationally strength trained participants. Furthering this to populations of powerlifters, it can be seen that their ILEX strength is similar to soccer players (figure 8). The greater ILEX SI in soccer players compared to the asymptomatic group might suggest soccer

is sufficient to strengthen the lumbar extensors, but considering typical strength based exercises do not improve ILEX torque (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013), and that soccer players ILEX torque is not different to strength trained participants and even competitive powerlifters, then this conclusion seems unlikely. Instead, the findings may reflect a selection bias, where stronger individuals are more likely to engage in sport and resistance training to begin with. The more notable finding is that soccer players, strength trained participants, and powerlifters all possess a similar level of ILEX strength, which suggests an inability to strengthen the muscle without pelvic restraints and is supported by experimental evidence (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013).

Another aim of this study was to assess whether lumbar extensor fatigue is possible after performing a soccer simulation, and to compare this magnitude to the magnitude of fatigue in the trunk flexors. Indeed, the principal finding from this study is that lumbar extensor fatigue can occur after performing soccer specific movements for 90 minutes. This is the first study to demonstrate that these muscles can be fatigued during sport-specific exercise without pelvic restraints. This has considerable implications for tasks requiring lumbar extensor force, such as maintaining sprint kinematics. Without access to equipment capable of strengthening this muscle, athletes might be susceptible to compensations from surrounding muscles leading to an earlier onset of fatigue (Raabe and Chaudhari 2016), changes to kinematics, and reductions in performance. Fatigue in the core muscles has been shown to decrease running time to exhaustion by ~4 minutes (Tong *et al.* 2014).

It could be argued that the reduction in lumbar force is not a result of mechanical work in these muscles. Supraspinal fatigue resulting from soccer would reduce lumbar extensor force regardless of any potential role in sprinting and movement. Indeed, supraspinal fatigue is evident after both competitive and simulated soccer, albeit the reduction in activation seems small (~5–10%; Brownstein *et al.* 2017; Thomas *et al.* 2017). It seems likely that isolated lumbar extension fatigue was reflective local fatigue, as the change was lower (reduced more) than the change in hand grip

strength. This provides evidence that local ILEX fatigue does occur after the SAFT90 and aligns with a recent meta-analysis that found no evidence of non-local muscle fatigue (Behm *et al.* 2021).

This study also compared the magnitude of trunk flexion fatigue to the magnitude of fatigue in the lumbar extensors. Baseline trunk flexion endurance time was less than observed in professional soccer players by ~28 s, also obtained using McGill's trunk flexor hold (Abdallah, Mohamed and Hegazy 2019). Whilst professionals may have better trunk flexion strength and endurance, the disparity in findings can also be attributed to the addition of a 5 kg weight and limiting hip flexor involvement in this study, indicative that these measures worked. This study was unable to reject the null hypothesis that the change in trunk flexion endurance time was greater than 0 (i.e. no change). The change in trunk flexion endurance was more varied than in the lumbar extensors (figure 10) but this was largely due to 1 individual recording a 50% increase in trunk flexion endurance time after performing the SAFT90. It's not entirely clear why this was the case, but the participant expressed that they frequently trained the trunk flexors by repeating a similar task. Thus, the individual could have unusually conditioned trunk flexor muscles that were unaffected by the SAFT90. Despite the greater variability, the median value for the change in trunk flexion is similar to the change in hand grip strength (figure 10), which suggests the participants who did experience a reduction in trunk flexion endurance did so because of non-local factors.

Unlike the trunk flexors, this study found that ILEX torque does reduce after the SAFT90 by 13%. The reduction in ILEX torque was remarkably similar to previous research where a 12% reduction in lumbar/thoracic extensor torque was found after soccer simulation (Fransson *et al.* 2018) suggesting that the pelvis was appropriately stabilised by Fransson *et al.* (2018) and adds confidence that the lumbar extensors are fatigued after soccer. Fransson *et al.* (2018) also found lumbar/thoracic extensor fatigue was greater than in the trunk flexors, which reduced by 10%. This is larger than the 4.5% change in trunk flexion endurance observed in this study. It is likely that this difference can be attributed to the modality of assessment. This study used a measure of trunk flexion endurance whereas Fransson *et al.* (2018) measured maximum torque. Using an isometric

trunk flexion endurance task to measure fatigue has the benefits of requiring little equipment and is easily implemented, but it seems to be less sensitive to changes in the force generating capacity compared to assessments of maximum torque. This is possibly because endurance tasks are more easily influenced by factors such as motivation. Nonetheless, taking the findings of this investigation and the findings of Fransson *et al.* (2018), it can be said with some confidence that lumbar extensor fatigue is greater than the fatigue in the trunk flexors after soccer simulation and that the lumbar extensor fatigue is because of local factors.

It was previously shown that in comparison to the other core muscles, the erector spinae (of which the lumbar extensors are a part of) are the most likely core muscle to be weak as they display a high level of activity during high-speed running (Saunders *et al.* 2005) due to the need to produce large torques when sprinting (Sado *et al.* 2019) relative to their capacity (Fransson *et al.* 2018). Considering this, and that it has been confirmed that this muscle experiences greater fatigue than the trunk flexors after soccer, then the erector spinae appears the most likely core muscle to increase the risk of HSIs due to weakness from soccer fatigue. This is particularly important as the lumbar extensors cannot be strengthened without restraining the pelvis (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013; Bruce-Low *et al.* 2012) and thus interventions designed to prevent fatigue in these muscles may not be accessible to some. Furthermore, interventions such as the FIFA11+ and those used by Sherry and Best (2004) provided exercises that focus on the anterior core muscles and carry the implicit suggestion that the anterior core muscles are most important, not the posterior core muscles. It is therefore imperative that any risk of HSIs that could arise from the magnitude of lumbar extensor fatigue after soccer is made clear.

It is anticipated that lumbar extensor fatigue will increase trunk lean during the stance phase of running as the lumbar spine cannot achieve the same degree of extension. In turn, this could lead to further anterior pelvic tilt, lengthening the hamstrings and increasing the risk of HSI. It is not known whether the magnitude of fatigue that is experienced after soccer is sufficient to induce these anticipated changes and it may be that surrounding muscles are be able to compensate

(Raabe and Chaudhari 2018). Thus, investigations should now seek to confirm whether this mechanism is realised with the magnitude of lumbar extensor fatigue measured after soccer.

There were some limitations with this study. Five minutes of passive rest was allocated between the completion of the simulation and the beginning of strength testing, which would remove the majority, if not all of the metabolic fatigue (Grgic *et al.* 2018). Periods of high intensity during match play will likely induce greater fatigue. Consequently, it is expected that the true reduction in ILEX torque and trunk flexion endurance is greater during match play compared to the measurements in this study. This delay was necessary to prepare the participants for the ILEX strength and trunk flexion endurance tests. Also, it is possible the repeated measures design meant participants altered their behaviour to meet the hypothesis. To counter this, it was ensured that all efforts were maximal using verbal encouragement and confirmation from participants that trials were indeed a maximal effort. It was also ensured that participants were unaware of their baseline torque or time for any of the measures. The follies of using soccer simulation instead of gameplay include assuming that this is a precise representation of the sport. However, it should be stressed that this estimate of trunk flexor, isolated lumbar extensor, and hand grip fatigue is not intended to reflect the typical fatigue after every match, but to demonstrate that over numerous games, the typical fatigue is likely to be near our estimate here.

3.1.6. Conclusion

It has been demonstrated for the first time that the SAFT90 protocol is capable of inducing ILEX fatigue and this is greater than the fatigue experienced in non-local muscles. It therefore seems likely that this will occur in real soccer match play, and possibly even to a greater extent if the demands exceed the simulation used here. This study failed to demonstrate that trunk flexor fatigue occurs after soccer simulation and doesn't appear to exceed the fatigue in non-local muscles during soccer. It has also been shown that the lumbar extensors are unlikely to be strengthened by playing soccer. Thus, it is imperative that the potential consequence of lumbar extensor fatigue from soccer match play on HSI risk are investigated.

3.2. REPLICATING SOCCER EQUIVALENT FATIGUE IN THE LUMBAR EXTENSORS USING ISOLATED RESISTANCE EXERCISE

3.2.1. Introduction

The previous study found the erector spinae and the encompassing lumbar extensors are fatigued during soccer match play which suggests they may become too weak for the demands of sprinting. It was hypothesised in section 2.4.3. that lumbar extensor fatigue will reduce the amount the lumbar extends during the stance phase of running, resulting in an increased trunk lean and subsequent anterior pelvic tilt. Thus, it is important the causal effects of soccer equivalent lumbar extensor fatigue on HSI risk are understood so that appropriate prevention programmes can be designed, as current interventions such as the FIFA11+ overlook the posterior core muscles (e.g. the lumbar extensors).

In order to ascertain the causal effects of lumbar extensor fatigue in soccer, the consistency assumption must be met (Hernán 2016). This means when examining the effects of lumbar extensor fatigue, the magnitude must be equivalent to that experienced in soccer without causing any other changes. Soccer simulation is clearly an inappropriate method for achieving this as it fatigues multiple muscles (Fransson *et al.* 2018; Greig *et al.* 2008), let alone a time-consuming task (105-minute duration). A commonly used exercise for investigating the effects of lumbar fatigue is the Biering-Sorensen task (Hart *et al.* 2009) but this possesses no association to ILEX torque (r = 0.06; Conway *et al.* 2016). Indeed, Hart *et al.* (2006) reported changes in the hamstring EMG median frequency after the Biering-Sorensen test indicating hip extensor fatigue. Instead, a device that restrains the pelvis such as that used by Bruce-Low *et al.* (2012) is appropriate for isolating the lumbar spine and has been used previously to induce lumbar extensor fatigue, albeit for much larger losses in force compared to that in soccer (Stuart *et al.* 2018). Though the ILEX device does limit work to the lumbar extensors, it cannot exclude an isometric contraction of the hip extensors as it attempts to posteriorly rotate the pelvis, which may violate the consistency assumption if a meaningful magnitude of fatigue occurred in these muscles. Though EMG studies indicate minimal

gluteus maximus activity (~12-15%; Udermann *et al.* 1999) and moderate hamstring activity (31-40%; San Juan *et al.* 2005) with pelvic restraints.

A plethora of studies have investigated the effects of soccer fatigue on injury risk (De Ste Croix *et al.* 2015; Lenhart, Thelen and Heiderscheit 2014; Greig and McNaughton 2014; Small *et al.* 2010— to name a few) but to our knowledge none have attempted to isolate the response of a single muscle or joint for its causal effects. Yet, if effective interventions are to be implemented then an understanding of which muscle should be targeted needs to be known. Furthermore, a protocol that replicates the ILEX fatigue from soccer can be used as a monitoring tool for clinicians to track changes to interventions and possibly monitor injury risk throughout a season. Similar methods for the lower limbs are used by practitioners (Pinto *et al.* 2017).

Therefore, the identification of such a protocol is key to ascertain the causal effects of lumbar extensor fatigue equivalent to soccer match play, and to potentially safeguard athletes from injury and other unknown effects. The purpose of this study was to create a lumbar extensor fatigue protocol, using a device that immobilises the pelvis, that is capable of replicating ILEX fatigue equivalent to soccer.

3.2.2. Method

3.2.2.1. Participants

Fourteen male amateur soccer players were recruited through convenience sampling (age: 20 ± 3 years; mass: 72.9 ± 10.1 kg; stature: 176.1 ± 6.9 cm). Seven players competed in college or university leagues and seven players competed between steps 9–15 (mode of 10) of the English national league system, and all players had outfield positions. Specifically, three were defenders, six were midfielders, and five were attackers. Participants had to be male due to differences in the fatigue response to a given lumbar fatigue protocol (Stuart *et al.* 2018) and were excluded if they were currently injured or experiencing any pain or soreness.

3.2.2.2. Procedure

To replicate the fatigue induced after a simulated football match, a protocol using an ILEX device (MedX, Ocala, FL) was designed using the results of Stuart *et al.* (2018). Participants were required to attend the university laboratory on three occasions. First, participants were familiarised with the procedure for maximum isometric lumbar extensor strength as per study 1. The second and third session, each separated by at least 72hrs, both began with a baseline measure of the ILEX strength across the full ROM (0°–72°) whilst at rest. After a ~1-minute passive rest, a dynamic fatiguing task was completed. The aim of this task was to induce a similar degree of fatigue experienced after completing a simulated football match (SAFT90 protocol), approximating a reduction in SI of 2767 N·m·deg.

The results of male participants in Stuart *et al.* (2018) were used to estimate the number of repetitions required to induce a similar degree of fatigue by assuming the change in SI was a linear function of time under tension in the 80% of peak torque load condition (high load). This assumption was based on the results of Gorostiaga *et al.* (2012) who found peak power decreased linearly with adenosine triphosphate reduction whereas the reductions in power with lactate followed a non-linear relationship. Thus, the 50% peak torque condition (low load) of Stuart *et al*. (2018) was not included in the estimate due its potential to upwardly bias the estimate of fatigue per repetition.

In the high load condition for males, the time under tension averaged 58 s equating to approximately 8.3 repetitions. Given that the high load condition had a mean reduction in SI of 6167 N·m·deg, the SI change per repetition is estimated to be 743 N·m·deg. Therefore, to achieve the desired reduction in SI of 2767 N·m·deg, approximately 3.7 repetitions, or 4 to the nearest integer, at 80% of peak torque should be performed. However, this is based on fatigue measured immediately post-exercise. For a more practical use of this protocol and to align with the measurement of fatigue after simulated football in Study 1, post-exercise torque was measured after five minutes of passive recovery. Thus, additional repetitions may be needed to ensure

sufficient fatigue remains after the recovery period. As the magnitude of torque recovery after five minutes is unknown, the precise number of additional repetitions required cannot be calculated.

To identify the optimal prescription of repetitions and replicate soccer equivalent magnitude of fatigue, participants were tasked with completing two sessions in a randomised order, using the 'RANDOM' function in Excel software (version 2016; Microsoft, Reading) to assign each participant to a condition. Depending on their condition assignment, participants completed either four or five repetitions in the first session with 80% of their peak torque to induce an estimated SI reduction of 2972 and 3715 N·m·deg respectively. It was anticipated that these values will decrease towards the target value of 2767 N·m·deg after a 5-minute recovery and therefore induce adequate fatigue. Peak torque has shown to largely correlate with SI values (r = 0.92–0.93) according to the results of Study 1 and Stuart *et al*. (2018) respectively, was considered a practical method for calculating relative loads. The second session utilised the same protocol using the remaining scheme of repetitions. In both conditions, the target repetition speed was 7 s long (2:1:4 ratio for concentric, isometric and eccentric respectively), though repetition duration increased with fatigue.

3.2.2.3. Data analysis

The required precision of the estimate of lumbar extensor fatigue from the fatiguing protocol was unavailable from current literature as research is yet to investigate the meaningful effects of lumbar extensor fatigue on lumbo-pelvic kinematics. Teng and Powers (2016) found a positive correlation between hip extensor strength and trunk flexion, where a 2 N·m·kg increase in hip extensor torque correlated with a 1° increase in trunk flexion (r = 0.55). Using this as an anchor for our estimate, the change in the lumbar extensor SI to produce a 1°change in trunk lean would be 7488 N·m·deg based on the trapezoidal estimate of the integral, where x is lumbar extensor torque:

$$\int_{0^\circ}^{72^\circ} f(x) d12^\circ \tag{4}$$

where peak torque is equal to 140 N·m (2 N·m·kg where sample mass is 70 kg) and produced at full flexion (72°), decreasing linearly to 70 N·m at full extension with a ratio of 2:1 (raw data of Stuart

et al. 2018). However, a change in SI of 7488 N·m·deg is excessive for the lumbar extensors as the raw data of Stuart *et al.* (2018) show that performing repetitions of isolated lumbar extension to failure reduce SI by 6167 and 9119 N·m·deg for high loads and light loads respectively. This estimates that to produce a 1° change in trunk flexion the lumbar extensors must act to volitional failure. This seems unrealistic and so the hip extensors are unlikely to be representative of the lumbar extensors. In circumstances where precision cannot be deduced, arbitrary recommendations such as an effect size of 0.2 have been suggested (Cook *et al.* 2018; Hislop *et al.* 2014). This effect size recommendation is a conservative estimate in the dearth of a-priori knowledge, but here the desired distribution of data is known. Thus, the agreement of the protocol was estimated based on the results of *Study 1* using a Z-score approach. The Z−score was preferred as *t* scores provide intervals for the mean statistic and not the proportion of data about the mean. Likewise, with small samples, *t* statistics can provide a broad range of values reflecting the uncertainty and this would lead to low power to detect similarity as a range of values could fit the interval.

3.2.2.4. Statistical analysis

A z-score of 0.68 about the mean lumbar extensor fatigue from soccer was used to determine the range of equivalence. This score was chosen as it is equivalent to ± 25% of the population about the mean, assuming a perfectly normal distribution, and therefore reflects the majority of the population responses with regards to soccer induced ILEX fatigue. The population SD of lumbar extensor fatigue after soccer simulation was estimated using a sample standard deviation with n−1 degrees of freedom. A z score of 0.68 ± the mean fatigue produced from the SAFT90 produced an interval of −1502 to −4032 N·m·deg.

The protocol was deemed to agree with soccer induced fatigue when the upper and lower bound of the 90% CI did not overlap with −1502 and −4032 N·m·deg respectively. A 90% CI was chosen as this is analogous to two, one-sided t-tests used in equivalence testing. The probability of achieving

fatigue greater than the limits of the CI is equal to 5% over infinite replication studies. Statistical analyses were performed using SPSS (Version 25; IBM Corp. 2019).

3.2.3. Results

Following a Shapiro-Wilks test for normality, it was revealed data for the four-rep protocol was not normal ($W = 0.745$; df = 14; $p = 0.01$) but change scores for the 5-repetition protocol was ($W = 0.971$; df = 14; $p = 0.886$). Descriptive statistics showed the four-rep protocol induced a median of −1009 N·m·deg (interquartile range: 1497 N·m·deg) whereas the five-rep protocol induced a mean change of −2686 ± 1703 N·m·deg. It was subsequently decided to not further test the 4-rep protocol due to the median value being outside of the acceptable range (−1502 and −4032 N·m·deg) and therefore of insufficient force loss. The 90% CIs for force loss after five repetitions was −1880 to −3492 N·m·deg and within the acceptable interval (−1502 and −4032 N·m·deg)

3.2.4. Discussion

The aim of this study was to identify a protocol capable of inducing lumbar extensor fatigue with a magnitude equivalent to that experienced in soccer. It has been shown that a 5-repetition protocol with a load of 80% of peak torque is capable of achieving such requirements. This is the first study to induce fatigue that is specific to the lumbar extensors while achieving a sport specific magnitude of fatigue and offers a viable approach for investigating the causal effects of soccer equivalent lumbar extensor fatigue, and for assessing the effectiveness of training protocols and player monitoring.

The addition of one repetition from four to five had an effect that was surprising. The force reduction between four and five repetitions was −1677, 60% of the fatigue induced from soccer simulation. This is unexpected given the lumbar extensors are primarily type one fibres (Cagnie *et al.* 2015). The lumbar extensors are clearly sensitive to relatively small changes in intensity of effort (i.e. proximity to momentary failure) and raises the question as to whether the fatigue response approximates a step function, where a threshold of physiological work leads to large increases in

torque loss (fatigue). It would be interesting to assess if lumbar extensor fatigue is present at half-time as Woods *et al.* (2004) identified the final third of both halves in soccer as periods of increased HSI risk. If lumbar extensor fatigue is not present by half time, it may only have a small role in the increased injury risk in the final third of each half, even if the lumbar extensor fatigue is shown to increase anterior pelvic tilt. Furthermore, the fact a single set of five repetitions at 80% of peak torque is sufficient to induce the same fatigue experienced in soccer may have implications for the scheduling of lumbar extensor testing and training. Practitioners should be careful not to implement lumbar extensor testing or training prior to physical activity as this may impair performance or worse, leave them susceptible to injury. Future research would benefit by understanding the time to recover the loss in torque from soccer match play.

Some limitations are acknowledged with this procedure. The repeated measures design means this study is susceptible to demand characteristics, but the two levels of study mitigates potential problems as participants were unaware which protocol was thought to be more successful, nor were they aware of the desired fatigue from the protocols. Therefore, it seems unlikely demand characteristics explain these findings. The mechanism of inducing lumbar extensor fatigue is likely different to that in soccer, where fatigue accumulates from 90 minutes of match play compared to the five repetitions of weighted lumbar extension used here. Despite this, the protocol still achieves the loss of lumbar extension force from soccer match play that ultimately may affect kinematics. Because of the sensitivity of the lumbar extensors to one additional repetition, future work would benefit by examining the effects of parameters such as load to achieve a more precise estimate.

3.2.5. Conclusion

The results of this study have shown that the lumbar extensor fatigue induced by soccer simulation can be replicated in isolation and with reasonable precision by performing five repetitions at 80% of peak torque. The development of this protocol can be used to assess training adaptations in soccer players, and more importantly the causal effects soccer equivalent lumbar extensor fatigue can now be assessed while minimising confounding from synergistic muscles. The following study

will investigate the effects of this protocol and thus soccer equivalent lumbar extensor fatigue on running kinematics and its potential to increase HSI risk. If it is shown to increase injury risk, this protocol may well serve as a predictive tool for assessing injury risk in future studies.

3.3. THE CAUSAL EFFECTS OF SOCCER EQUIVALENT LUMBAR EXTENSOR FATIGUE ON RUNNING KINEMATICS

3.3.1. Introduction

Core strengthening is a common feature of hamstring injury prevention measures (Meurer, Silva and Baroni 2017; McCall, Dupont and Ekstrand 2016) but supporting literature is weak. A 2018 systematic review remarked at the absence of prospective studies examining pelvis and trunk kinematics and injury risk (Ceyssens *et al.* 2019). Nonetheless, a prospective empirical study has shown increased HSI risk with deviances in lumbo-pelvic kinematics whilst sprinting (Schuermans *et al.* 2017a). Specifically, an increase anterior pelvic tilt was found (Schuermans *et al.* 2017a) which is thought to strain the hamstrings further whilst running and therefore increase stress and the injury risk. Research is yet to identify the core muscles that are responsible for anterior pelvic tilt during running, but it is not without trying (Schuermans *et al.* 2017b; Sherry and Best 2004). There is some evidence suggesting erector spinae weakness is responsible (Bonte *et al.* 2015) but it is far from certain. Therefore, approaches to core strengthening for hamstring injury prevention lack precision.

The problem with this is that other training methods, such as increasing eccentric strength, are proven to be effective in reducing injury risk (van der Horst *et al.* 2018; Seagrave *et al.* 2014; Petersen *et al.* 2011) and other adaptations such as increasing fascicle lengths also show promise (Timmins *et al.* 2016). Therefore, a holistic core strengthening approach is performed at the cost of other preventative exercises, which may be superior. Indeed, eccentric strengthening seems to benefit from increased volume (Severo-Silveira *et al.* 2018). Likewise, the increase in fatigue from unnecessary exercise can limit adaptations and impair sensory learning (Branscheidt *et al.* 2019) or worse place athletes at greater risk of injury, as suggested by a recent increase in training injury rates (Eirale 2018).

The lumbar extensors undergo a large increase in activity during the stance phase in order to extend the lumbar spine (Saunders *et al.* 2005), and likely limit the trunk lean that occurs at this time (Schache *et al.* 1999). It was suggested in section 2.4.3. that failure to extend the lumbar spine would increase the trunk lean, and potentially increase anterior pelvic tilt (Higashihara *et al.* 2015). It was proposed the increased anterior pelvic tilt might arise from greater passive forces in the lumbar extensors as it is more flexed, but also by increasing the demands of the hip extensors to decelerate the trunk lean, resulting in less time for the hip extensors to achieve the same magnitude of posterior pelvic tilt, and thus the pelvis is more anteriorly tilted relative to normal running.

As these changes are expected to occur during stance, it could be argued injury risk will not increase as the swing phase appears the most likely time for HSI (Wan *et al.* 2017; Chumanov, Heiderscheit and Thelen 2011; Thelen *et al.* 2005a) but as mentioned previously, the phase of the stride cycle with the most risk in normal running conditions may not be the same as during runs that consequent in injury. Furthermore, changes to joint angles such as anterior pelvic tilt and hip flexion during stance could carry over to the swing phase of sprinting and increase hamstring lengths and thus stress in the more injurious phase.

Although a possible mechanism exists for lumbar extensor fatigue to increase HSI risk, it remains unknown whether the typical lumbar fatigue experienced in soccer is sufficient for increased anterior pelvic tilt to materialise. If it were, this would present considerable implications for practitioners and athletes, as specific training would be required to optimise hamstring injury prevention, which is only possible through pelvic restraints. Therefore, devices that are accessible may need to be developed. If soccer equivalent lumbar extensor fatigue were found to have no effect, this too would be of great concern as the potency of the surrounding core muscles such as the RA to also have an effect would be questionable based on their lower levels of activity during running (Saunders *et al.* 2018) and lesser fatigue after soccer (indicated by study 1 and Fransson *et al.* 2018). Thus, prevention programmes could focus on more effective strategies.

The aim of this study is to investigate running kinematics in amateur soccer players before and after an ILEX fatiguing protocol that induces fatigue equivalent to that experienced in soccer. It is hypothesised that soccer equivalent lumbar extensor fatigue increases anterior pelvic tilt during the stance phase of running and that these effects will continue to be present in the swing phase.

3.3.2. Method

3.3.2.1. Pilot study

To answer the aims of this study, a pilot study was carried out to ensure the most externally valid design feasible was used. A repeated measures design was used to compare 3-dimensional overground sprint kinematics before and after fatiguing the lumbar extensors to a magnitude equivalent in soccer. The independent variable was the condition (pre and post lumbar fatigue) and the dependent variables were joint angles for the lumbar spine, pelvis, hip, and knee in the sagittal plane.

A convenience sample of 14 amateur soccer players were recruited (Age: 21 ± 3 years; Mass: 74.3 ± 11.3 kg; Stature: 177.3 ± 7.0 cm). Participants were excluded if they were currently injured or experiencing any pain or soreness. Sample size estimates were obtained through a power analysis of prior research. Visual inspection of data for soccer players at risk of hamstring injury showed an effect size of 13° for anterior pelvic tilt (Schuermans *et al.* 2017a) whereas soccer simulation resulted in an effect size of 4.96° for anterior pelvic tilt (Small *et al.* 2009). Due to the multifactorial nature of anterior tilt, the effects reported by these studies are likely to be larger than when considering the lumbar extensors alone. Due to the novelty of this area of research, a more conservative effect of 0.7 (angle change of ~3°) was chosen based on logistical considerations (e.g. participant recruitment). This required an estimated 15 participants to achieve a power of 0.8 and an alpha of 0.05 for a one-sided paired t-test.

Sprint trials took place indoors on rubber flooring and the lumbar fatigue protocol took place in a temperature-controlled laboratory. Three sprint trials were performed at baseline and these were

repeated after completing a lumbar extensor fatiguing protocol shown to replicate the typical fatigue after soccer simulation (study 2). An 8-camera set up (MX T20, Vicon, Oxford) was used to capture the sprints in 3-dimensions at 250 hz. The sprint track was 30 m long with the origin of the capture volume (6 m x 6 m x 2 m) at 15 m, allowing for a further 5 m of sprinting and an additional 4 m for deceleration after capture. Cameras detected retroreflective markers attached to the subject in the 2-dimensional space and calculated their respective three-dimensional coordinates in the global reference frame.

Unfortunately, this design had logistical problems that reduced the quality of data. As an example, the coefficient of variation for the change in lumbar flexion was 57%. It was suspected that the reason for the large variability was the indoor venue, which had reflections that required camera masking using software (Vicon, Nexus version 2.8) and led to gaps in the captured trials. As a result, there was often only one suitable trial captured for each condition despite the three attempted trials. Finally, the time taken to reach the indoor venue and re-attach the markers after completing the lumbar extensor fatiguing protocol was 12 minutes. This is considerably longer than the five-minute pause used to replicate soccer equivalent ILEX fatigue and It was expected the magnitude of ILEX fatigue had reduced below the target amount at the commencement of the post sprint trials. Thus, to answer the research questions, a design that possessed more internal validity was required.

3.3.2.2. Study design

A repeated measures design was used to determine the effects of soccer-equivalent ILEX fatigue on treadmill running kinematics. Treadmill running has been shown to produce similar kinematics to overground running (Hooren *et al.* 2019; Schache *et al.* 2001) and was therefore a suitable alternative to overground sprinting. Treadmill running trials and ILEX fatiguing exercise took place in a temperature-controlled laboratory. The independent variable was the condition (pre and post lumbar fatigue) and the dependent variables were the sagittal plane joint angles for the lumbar spine, pelvis, hip and knee. All testing was conducted during the off-season.

3.3.2.3. Participants

A convenience sample of 14 amateur soccer players were recruited (age: 22 ± 5 years; mass: 75.2 ± 10.6 kg; stature: 178.5 ± 6.8 cm). Eleven of the soccer players competed between steps 3–15 in the English national league system (mode was level 4) and three of the players competed in a university or college league. The sample consisted of three defenders, seven midfielders, and four attackers. Participants were excluded if they were currently injured or experiencing any pain or soreness. Participants had to be male due to differences in the fatigue response to a given lumbar fatigue protocol (Stuart *et al*. 2018) and must compete as an outfield player. The required sample size was identical to that in the pilot study (15 participants) based on a power analysis to detect an effect size of 0.7 (Cohen's *d)* equivalent to pelvis angle change of ~3°

3.3.2.4. Procedure

All running trials were performed on a motorised treadmill to standardise the running speed, which was set to the maximum speed of 24 km/h (6.66 $m \cdot s^{-1}$). To estimate the relative effort of treadmill sprinting, participants performed two maximum sprint trials on a non-motorised treadmill for convenience (Curve, Woodway, WI). Participants were first familiarised with the non-motorised treadmill and then performed two maximum sprint trials in an effort to achieve the highest speed possible. The greatest speed recorded was used as an estimate of the participants' maximum sprint speed.

Joint kinematics were captured during running trials on a motorised treadmill (260G, PulseFitness, England) using three-dimensional motion capture. Participants were familiarised with the running speed until they reported being comfortable. After a rest period, participants ran for 5 s to capture at least 16 strides (assuming a step frequency of 3.2 Hz; Nagahara *et al.* 2018). Recording and analysing multiple strides allowed a more accurate estimate of the true subject joint angles and reduced the inter-subject SD (Baker *et al.* 2019). Further strides were not recorded to limit the effects of fatigue. The lumbar fatigue protocol was performed as per study 2. Briefly, participants performed five repetitions of isolated lumbar extensions using 80% of peak torque (MedX, Ocala,

FL). All participants were familiarised with the device prior to testing. Post fatigue sprint trials followed the same method as baseline trials and were completed after precisely five minutes of passive rest.

3.3.2.5. Biomechanical model

Following the lower body Plug-In gait model (Vicon, Nexus version 2.8), sixteen retroreflective markers (14 mm) were placed on the lower limbs and pelvis and secured using double sided carpet tape. The three marker cluster used to model the lumbar spine as per Steele *et al.* (2014) and Schache *et al.* (2002) was placed perpendicular to the 12_{th} thoracic vertebrae (T12) to produce a local reference frame at the lumbar spine and create a rigid body model. The lumbar spine was modelled as a child segment from the pelvis (the parent segment). Previous research has shown this marker to have good within-day reliability in running (Schache *et al.* 2002) but between-day reliability was susceptible to errors from marker placement (Schache *et al.* 2002). To prevent this potential error, the location of retroreflective markers were marked using indelible pen for consistency upon reapplication after the ILEX fatiguing exercise. The locations of the retroreflective markers for the anterior superior iliac spine, posterior superior iliac spine, and femur were also marked.

3.3.2.6. Camera set-up

An 8-camera set up (MX T20, Vicon, Oxford) was used to capture the treadmill runs in three-dimensions. Cameras detected retroreflective, 14 mm markers attached to the subject in the two-dimensional space and calculated their respective three-dimensional coordinates in the global reference frame. Cameras were aimed to maximise coverage of the participant on the treadmill with five of the cameras mounted on the wall and the remaining three positioned on tripods around the front of the treadmill to capture the anterior pelvis markers (figure 11). All cameras recorded at 250 Hz. This was to limit reflective artefacts and satisfies the requirements of the Nyquist sampling theorem, as the pelvis frequency (the key joint angle for inferring HSI risk) is approximately 4 Hz when sprinting (Nagahara, *et al.* 2017). Image errors were less than 0.3 and in alignment with manufacturer guidelines.

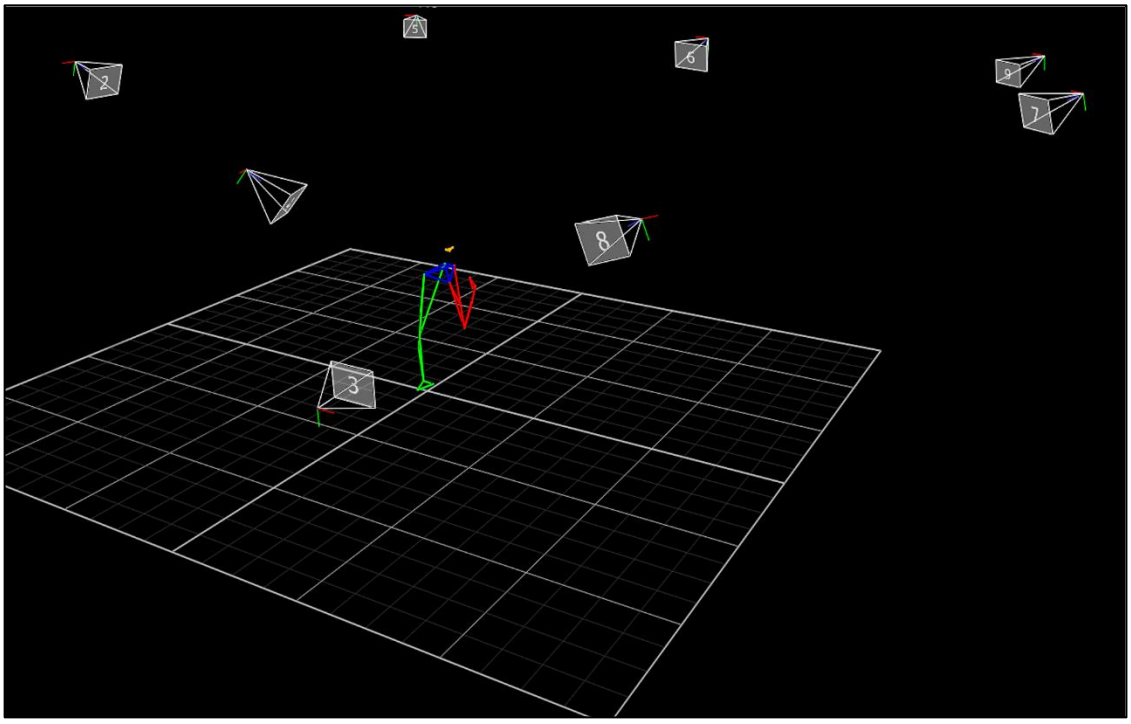

Figure 11: Position of the cameras around the participant running on the treadmill. Cameras are labelled 2 –9 because camera 1 was not capturing data due to a known technical fault.

3.3.3. Data analysis

The initial four and final four strides were removed for pre and post running trials to limit acclimatising and anticipatory effects of treadmill running. For the remaining strides, gaps were filled in the order of spline fill (up to 12 frames), rigid body fill for the pelvis (up to 62 frames), and

pattern fill for all remaining gaps (up to 25 frames). Trajectories were then filtered using Woltering spline smoothing (tolerance of 10 mm^2). All processing was performed in Vicon Nexus software (version 2.8; Vicon, Oxford). Pelvis, hip, and knee angles were calculated using the lower body Plug-in Gait model (Nexus 2.8, Vicon, Oxford). Lumbar spine joint angles were calculated using a custom Bodybuilder (Vicon, Oxford) code pipeline used by Steele *et al.* (2014) and Schache *et al.* (2002) and was calculated as Cardan (Eular) angles and rotated in the x-y-z sequence.

Foot strike was defined as the instance of positive vertical velocity in the heel or toe marker for heel strike or forefoot strike respectively (confirmed visually; Milner and Paquette 2015), and toe-off was defined as the moment of peak knee extension after foot strike (Fellin *et al.* 2010). The stance phase was defined as the time between foot strike and toe-off, and the swing phase was defined as the time between peak knee flexion after toe-off to peak knee extension. The time between toe-off and the start of swing was referred to as the pre-swing phase.

For each subject, joint angles for peak anterior and posterior pelvic tilt, and peak lumbar extension and flexion were calculated in the stance and swing phases for each recorded stride in the pre and post ILEX conditions. Joint angles from each stride were then averaged in the dominant and non-dominant side in each condition. Differences between joint angles after ILEX fatigue were compared for the dominant and non-dominant sides separately, as kinematics differ between dominant and non-dominant limbs (Haugen *et al.* 2017). However, the change in each limb after fatigue is expected to be homogenous, so the change in angle for left and right sides were averaged again to create a single value for the change in angle per subject and per phase. All joint angles were processed using Excel (version 2016; Microsoft, Reading).

3.3.4. Statistical analysis

Statistical analyses were performed using JASP (Version 0.14.1). According to a Shapiro-Wilk's test, data for the change in joint angles were normally distributed apart from the change in peak lumbar extension and flexion during swing (W = 0.782; p = 0.03 and W = 0.849; p = 0.02 respectively).

Therefore, lumbar flexion and extension during swing were described using medians and interquartile ranges, whereas all other changes were described using means and SDs.

The hypothesis that lumbar extensor fatigue causes reduced lumbar extension and increased anterior pelvic tilt was tested using one-sided, one-sample t-tests for the change in peak anterior and posterior pelvic tilt, and the change in peak lumbar flexion and extension during stance. If these were statistically significant, then follow up tests on the same joint angles were conducted during the swing phase to test the hypothesis that these changes are carried over to the swing phase of running. This was conducted using a one-sided, one sample t-test for the pelvis, or a one-sided Wilcoxon signed rank for the lumbar spine. All tests had an alpha of 0.05.

As part of exploratory analyses, the absolute lumbar angle in the global reference frame (sagittal lumbar angle + sagittal pelvis angle) was calculated, as a measure of the lumbar spine's orientation (i.e. forward lean). The absolute lumbar angle, along with the sagittal joint angles for the lumbar spine, pelvis, hip, and knee were time normalised to one stride cycle and visually presented. Joint angles in the lumbar fatigue condition were then compared to joint angles at baseline across the gait cycle using statistical parametric mapping one-sided paired samples t-test (SPM{t}), with an alpha of 0.05. Statistical parametric mapping allows inferences of continuous data without inflating the error rate and are now common in analyses of biomechanical data (Nüesch *et al.* 2019; Schuermans *et al.* 2017a). This technique means potentially important information is not lost by analysing discrete data only. All SPM analyses were performed in MatLab (R2021a) using open-source code available at SPM1D (https://spm1d.org/).

3.3.5. Results

The mean top speed whilst running on a non-motorised treadmill was 24.6 ± 2 km/h (6.83 $m \cdot s^{-1}$). Therefore, participants ran close to 100% of their maximum sprint speed. Non-motorised treadmill speeds are expected to underestimate over-ground speed due to belt friction so this should be considered an approximation of sprint speed.

Descriptive statistics for the change in lumbar spine and pelvis joint angles are presented in table 9. One subject was missing pelvis data during the post fatigue trials, therefore pelvis and lumbar comparisons were made with 13 subjects. All other comparisons were made with all 14 subjects. During the stance phase of treadmill running, inferential statistics failed to reject the null hypothesis that there is no increase in anterior pelvic tilt or lumbar flexion at the moment of peak anterior pelvic tilt, peak lumbar flexion, and peak lumbar extension ($p > 0.05$). At the moment of peak posterior tilt, inferential statistics rejected the null hypothesis that there is no increase in anterior pelvic tilt here ($t_{(12)} = 3.296$; $p = 0.03$). The mean increase in anterior pelvic tilt at this moment was 1.2° ± 1.3°. Follow up tests were conducted during the swing phase where it was found that the pelvis remained more anteriorly tilted at the moment of peak posterior pelvic tilt ($t_{(12)} = 2.398$; $p = 0.017$), with a mean increase of 1.3° ± 2.0°.

Table 9: Change in running joint angles with ILEX fatigue

Phase	Change in pelvis at peak anterior tilt	Change in pelvis at peak posterior tilt	Change in lumbar at peak extension	Change in lumbar at peak flexion
Stance	0.5° ± 2.6°	1.2° ± 1.3° †	0.4° ± 2.2°	0.6° ± 2.9°
Swing	0.6° ± 3.2°	1.3° ± 2.0° †	0.3° (2.0°)	0.8° (2.3°)

Note: Positive values indicate an increase in anterior tilt and lumbar flexion. Data are presented as means ± SDs apart from lumbar angles during swing which are medians and interquartile ranges. †Significant increase.

Exploratory analyses considered whether angles at any other phase in the gait cycle were altered (figure 12). SPM{t} inferential statistics showed that the angle of the lumbar spine relative to the laboratory reference frame increased just after toe-off until the start of swing (34–54% of stride cycle; figure 13) with a maximum difference of 2.6°, yet the lumbar spine relative to the pelvis was unchanged throughout the stride cycle (figure 13). Soon after the lumbar had become more inclined in the laboratory reference frame, the pelvis became more anteriorly tilted (51%—57% of stride

cycle; figure 14) by a maximum of 1.5°. Later into the swing phase the hip was more flexed for a brief moment (88%–91% of the gait cycle; figure 14) with a maximum increase of 2.9°. In addition, the pelvis became more anteriorly tilted at the terminal swing phase (97%–100% of stride cycle; figure 14) by a maximum of 1.4°. SPM{t} identified no changes at the knee across the gait cycle.

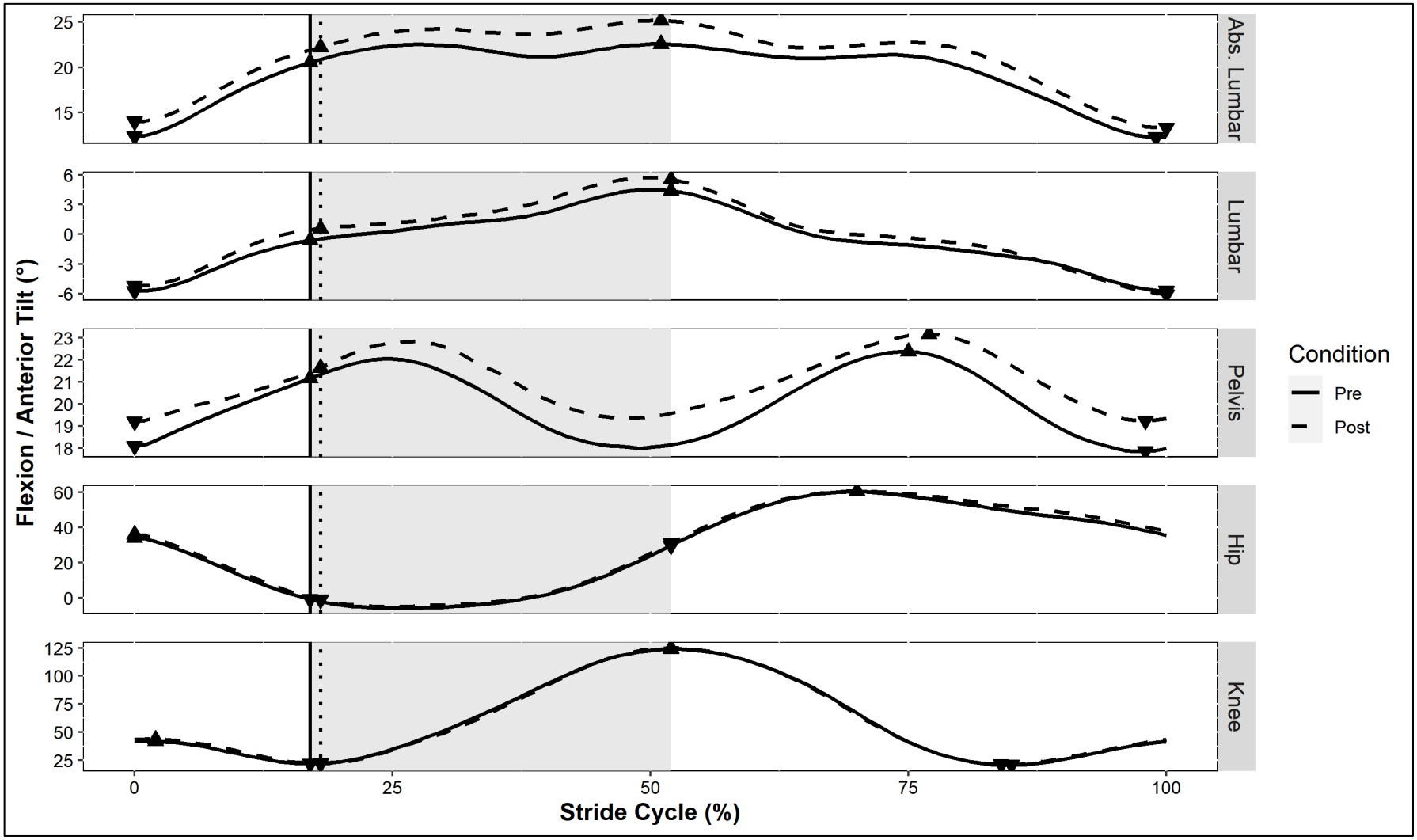

Figure 12: Kinematic waveforms of the joint angles for the absolute lumbar spine, relative lumbar spine, pelvis, hip, and knee across the stride cycle. Solid and dashed line represent toe-off for pre and post conditions respectively. Grey shaded area represents pre-swing phase.

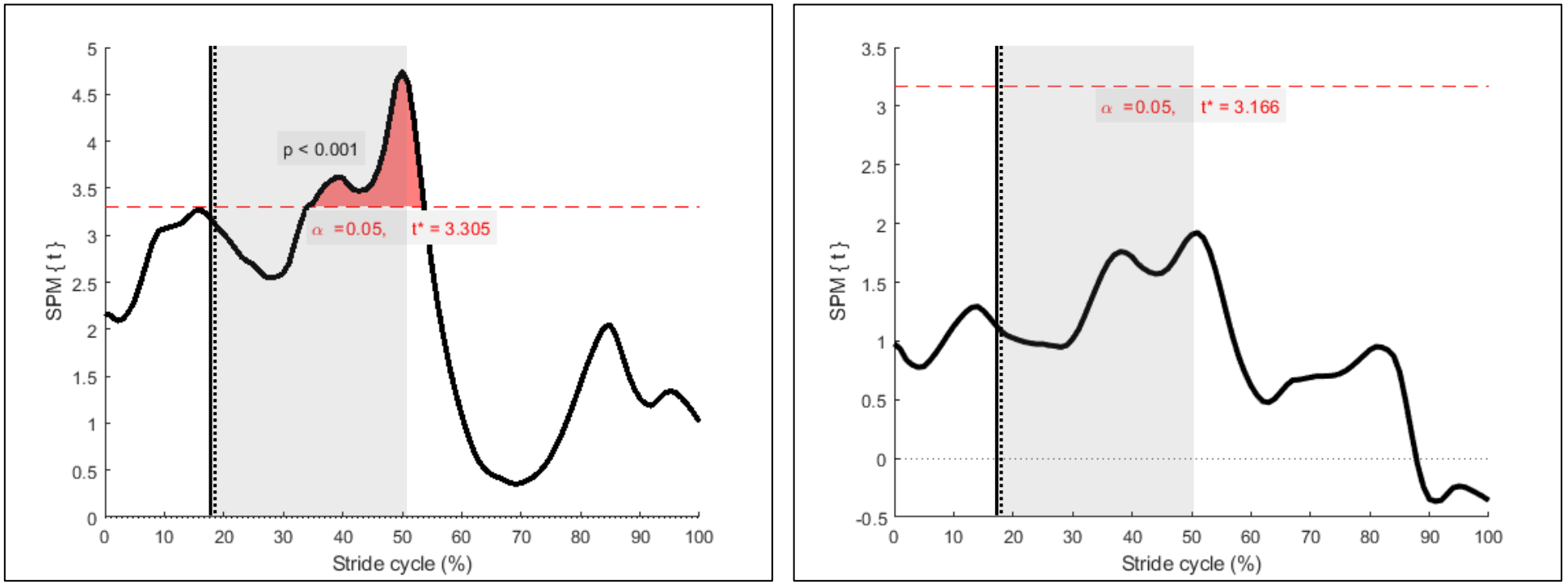

Figure 13: SPM[t} of the paired samples t-test of the absolute lumbar spine angle (left) and the relative lumbar spine angle (right). Areas shaded red show statistically significant increases in lumbar lean. Solid and dashed line represent toe-off for pre and post conditions respectively. Grey shaded area represents pre-swing phase.

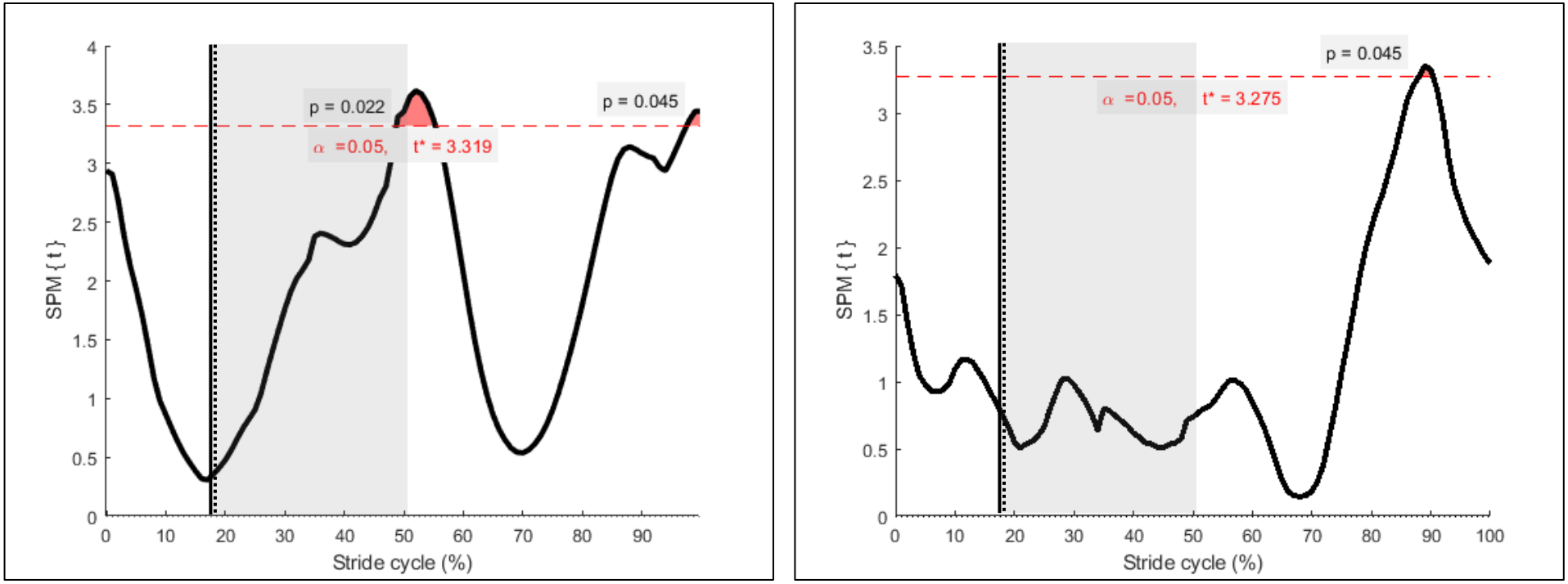

Figure 14: SPM[t} of the paired samples t-test of the pelvis angle (left) and the hip angle (right). Areas shaded red show statistically significant increases in anterior pelvic tilt and hip flexion respectively. Solid and dashed line represent toe-off for pre and post conditions respectively. Grey shaded area represents pre-swing phase.

3.3.6. Discussion

The aim of this study was to identify whether soccer-specific lumbar extensor fatigue increases anterior pelvic tilt and therefore the risk of HSI. It was hypothesised that the lumbar extensor fatigue would reduce the amount of lumbar extension, increasing the forward lean and the subsequent anterior pelvic tilt. It was expected that this would occur during the stance phase of running and carry over to the swing phase.

The investigation failed to identify an increase in peak anterior tilt or lumbar flexion during the entire stride cycle. However, peak posterior pelvic tilt was reduced (more anteriorly tilted) during the ILEX fatigued running trials. This might be because of the increased hip extensor demands that are expected to occur with forward lean, preventing the hip extensors ability to posteriorly tilt the pelvis. Exploratory analyses also showed a reduction in posterior pelvic tilt (more anteriorly tilted) during the terminal swing phase (figure 14 and table 9) and of similar magnitude (1.2° ± 1.3° and 1.3° ± 2.0° respectively). It is also notable that the increase in anterior tilt at the moment of peak posterior tilt at touch-down was not identified by SPM. This may be because discrete comparisons of joint angles were performed on data that was not time normalised. The SPM{t} value was high for a similar period but did not achieve the SPM{t} threshold of 3.319. Though at terminal swing, the reduced posterior pelvic tilt was also identified by SPM suggesting this is a more robust finding compared to the reduced posterior tilt at touch down.

Though the pelvis was found to be more anteriorly tilted at portions of the running stride (table 9 and figure 14), the lumbar spine was found not to flex relative to the pelvis throughout the stride. This is contrary to expectation, as reduced lumbar extension was suspected as the root cause of increased anterior pelvic tilt in the presence of lumbar extensor fatigue. One explanation is that if the pelvis did anteriorly tilt as the lumbar spine flexed, then the relative angle of the lumbar spine would appear unchanged or be of very small magnitude. Indeed, the lumbar spine angle relative to the laboratory reference frame (lumbar spine lean) was found to increase in exploratory analyses (figure 13). Of course, this increase in lumbar lean could have resulted from anterior pelvic tilt,

which would incline the lumbar spine, but this seems unlikely as the peak magnitude of change in the absolute lumbar spine angle was greater than the peak magnitude of change in the pelvis (2.6° and 1.5° respectively). Furthermore, the increase in lumbar lean identified by SPM{t} occurred prior to the increase in anterior pelvic tilt by 17% of the stride cycle suggesting the lumbar led the pelvis rather than the pelvis leading the lumbar spine. Whilst the temporal connection between the two joint angles does not prove causality, it is consistent with the original hypothesis.

The hip was found to be more flexed during the terminal swing phase for a brief moment (3% of stride cycle). This might be indicative of hip extensor fatigue that arose as a side-effect of the lumbar extensor fatiguing protocol. While hip extensor activity is small during ILEX exercise, it is not removed completely (San Juan et al. 2005; Udermann et al. 1999). Though if hip extensor weakness had occurred, one would expect to find changes in hip extension during stance too, as hip extensor torques are greatest during stance at the speeds used in this study (Schache *et al.* 2011). This was not observed. Furthermore, the largest hip torques during swing are eccentric, whilst the hip is flexing (Schache *et al.* 2011) but here the differences occurred whilst the hip had already begun extending (figure 12).

The results of (Schuermans *et al.* 2017a) show hamstring injured participants had more anterior pelvic tilt at the end of backswing (the period between toe-off and hip extension), which is similar to the findings here where the pelvis was more anteriorly tilted just after hip extension but before the knee had begun to extend into forward swing. Schuermans *et al.* (2017b) was unable to identify a core muscle that might have been responsible, but this study supports the notion the erector spinae, and its lumbar extensors, are at least partly responsible. Though Schuermans *et al.* (2017a) reported injured subjects had a ~13° more anteriorly rotated pelvis (obtained via WebPlotDigitiser; Rohatgi 2019). In this study we observed a peak difference of 1.5° during this phase, just 11% of the change observed by Schuermans *et al.* (2017a). This can be partly explained by the fact Schuermans *et al.* (2017a) trials were recorded at 15–25 m of sprinting and in a subset of soccer players (prospectively hamstring injured players). Injured soccer players, such as those used by Schuermans

et al. (2017a), might be more susceptible to erector spinae weakness, representing a subset of soccer players that might have shown more extreme responses to erector spinae fatigue in this study. Prospectively comparing baseline ILEX torque between uninjured and subsequently injured soccer players may shed light on this topic but could require many participants and thus substantial time to recruit a pool of subjects who have the same cause of injury (i.e. erector spinae weakness). In figure 3, it is shown that there are many paths to injury which would confound any attempted association.

Bonte *et al.* (2015), who observed lower erector spinae activity in injured subjects, found increased anterior pelvic tilt at the moment of foot strike, which broadly agrees with the changes observed in this study with ILEX fatigue. However, Bonte *et al.* (2015) attributed the increased pelvic tilt at touchdown to increased forward lean in the prior swing phase, yet this study did not find an increase in lumbar lean in terminal swing. It is worth noting that the increase in lumbar lean observed during pre-swing (34–54% of stride cycle) is expected to coincide with terminal swing on the contralateral limb but it's unclear why the same finding would not be repeated in the ipsilateral limb. The findings of Schuermans *et al.* (2017a) produced the same issue, where anterior pelvic tilt increased during back swing, coinciding with the swing phase on the contralateral limb, but no change in pelvic tilt during swing in the ipsilateral limb. Future research might benefit by using non-cyclic tasks such as a broad jump to understand if changes can be attributed to stance or swing.

The data in this study indicates that lumbar extensor fatigue equivalent to soccer would lengthen the hamstrings via anterior pelvic tilt during the pre-swing phase (as the contralateral limb is in terminal swing). Thus, the findings of this study would suggest that, in the strict causal sense, lumbar extensor fatigue increases hamstring injury risk. Though it may only play a small part in the injury mechanism as anterior pelvic tilt was increased by no more than 1.5° (peak difference between means). With that said, it may be that a subset of soccer players experiences more drastic changes because of lower baseline erector spinae weakness. Further to this, this study induced lumbar extensor fatigue that was equivalent to the fatigue in soccer measured five minutes after

passive rest. Lumbar extensor fatigue during match play may well be greater and causing larger changes that those observed here.

The lumbar extensor portion of the erector spinae might be problematic for hamstring injury prevention as weakness cannot be addressed through typical strength training methods, as a system restricting pelvis motion would be necessary (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013; Bruce-Low *et al.* 2012). Thus, without special consideration, lumbar extensor weakness would remain a persistent risk even with typical strength training measures. Fortunately, it seems that for the average soccer player, the increase in risk might be small (1.5° increase in anterior pelvic tilt) and time might be better utilised performing exercises such as the Nordic curl, which has proven reductions in injury rates (Petersen *et al.* 2011). Though at an individual level, if an athlete was displaying an increase in forward lean during running it may be worth considering the lumbar extensor strength if access to pelvis restraints are available.

The ILEX device restricts mechanical work to the lumbar extensors, but it cannot avoid an isometric contraction of the hip extensors as it attempts to posteriorly rotate the pelvis. Nonetheless, EMG studies indicate minimal gluteus maximus activity (~12-15%) and submaximal hamstring activity (31-40%; San Juan *et al.* 2005; Udermann *et al.* 1999). Furthermore, their action is limited to isometric contractions for 15 s (upward phase). It seems unlikely that a 15 s submaximal isometric task would produce any considerable hamstring fatigue that persists after five minutes of rest. The analysis revealed no changes at the hip during periods where hip extensor torques are high (stance phase and hip flexion during swing) indicating a reduction in hip extension torque was not responsible but this requires confirmation. The repeated measures design means demand characteristics could be a factor. Participants were informed that the study was examining changes in sprint technique after fatigue, but no precision was given as to which changes were under investigation and may have been interpret broadly. If demand characteristics were a factor, the lack of precision means any effect will be randomly distributed among samples and add noise that will statistically be ignored.

3.3.7. Conclusion

This study set out to test whether soccer-equivalent lumbar extensor fatigue could alter kinematics and increase HSI risk. The results here show the pelvis becomes anteriorly rotated at the start of swing and again during terminal swing, which carries into foot strike. With no other changes at the hip and knee, this would lengthen the hamstrings and increase injury risk according to the model of injury in figure 3, albeit the increase in risk would appear small.

3.4. THE CAUSAL EFFECTS OF SOCCER EQUIVALENT LUMBAR EXTENSOR FATIGUE ON HAMSTRING TORQUE

3.4.1. Introduction

In the previous study it was demonstrated that lumbar extensor fatigue from soccer can increase the risk of hamstring strain injury by inducing anterior pelvic tilt. Unfortunately, the lumbar extensors are not easily strengthened (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013) and preventative measures such as hamstring strengthening exercises may be the most appropriate method of reducing the risk arising from lumbar extensor fatigue. The Nordic curl has been frequently trialled with great success in preventing injuries (van Dyk, Behan and Whiteley 2019), and it's expected this is partly due to increases in biceps femoris fascicle length (Bourne *et al.* 2018; Timmins, *et al.* 2016).

Hamstring actions with high intensity appear necessary to Increase fascicle lengths (Bourne *et al.* 2018). Specifically, high forces are needed rather than high levels of muscle activity as eccentric modalities perform better than concentric modalities (Bourne *et al.* 2018), especially at long muscle lengths (Guex *et al.* 2016). Lovell *et al.* (2018) found Nordic curls performed after soccer led to no change in fascicle lengths yet performed before soccer there were large increases, further suggesting the need for high forces rather than activity. Lumbar extensor fatigue after soccer has the potential to reduce hamstring forces during the Nordic curl and could partly explain why Lovell *et al.* (2018) observed no increase in FLs.

Sado (2016) suggested large hip extensor forces would need to be opposed by the lumbar extensors to maintain the pelvis angle. This did not appear to be responsible for the peak in erector spinae activity during running due to the difference in timing between peak hip extensor force and peak erector spinae activity (indicated by Saunders *et al.* 2005 and Schache *et al.* 2011). When hip extensor actions are more forceful, such as those in slower actions used for training and testing, fatigue in the lumbar extensors may render them unable to oppose the hip extensors pull, resulting

in a more posteriorly tilted pelvis. This would result in a shorter muscle-tendon length for a given knee or hip angle and in turn produce less passive force at longer muscle lengths.

Schuermans, van Tiggelen and Witvrouw (2017) observed delayed hamstring onset in prospectively injured subjects, causing hip extension to be initiated by the erector spinae. They proposed the injured participants are at greater risk because the hamstring would be 'deprived of sufficient training stimuli' at the beginning of actions. In the same sense, if lumbar extensor weakness lowers the forces during hamstring actions, it too might deprive the hamstring of the training stimuli. Particularly as participants engage in long term interventions and develop hamstring strength, the limit to their development could lie in the lumbar extensors ability to oppose its rotation on the pelvis.

Narouei (2018) claimed erector spinae and multifidus muscle activity is greater than any other trunk muscle during the Nordic curl exercise, second only to the hamstrings. Though without normalisation to a maximal contraction, comparisons between muscles are not possible. Even so, if erector spinae activity was found to be greater, it might be to resist the gravitational moment that acts on the trunk rather than the hamstring torque posteriorly tilting pelvis. This doesn't appear to be the case as when anterior trunk displacement is increased during the Nordic curl by using a 10° and 15° downward slope, erector spinae activity remains unchanged (43 ± 12% of maximum at 10° compared to 43 ± 14% of maximum at 15° of slope; $p > 0.05$). In contrast, when the knee is extended during the Nordic curl by the same magnitude (10° to 15° of extension), increasing the hamstring torque, the erector spinae activity increased from 47 ± 14% to 56 ± 23% of maximum ($p < 0.05$), suggesting the high erector spinae activity during the Nordic curl is in response to the hamstring torque rather than the gravitational trunk flexion moment (Park, Kim and Park 2019). Further, when comparing descriptive statistics, the prone leg curl exercise with the trunk supported, achieved greater ES activity than the Romanian deadlift and good morning exercise where the hip is flexed and the trunk unsupported (McAllister *et al.* 2014). Clearly erector spinae activity increases in response to hamstring torque. If the erector spinae become fatigued or cannot be strengthened

alongside the hamstrings, the hamstring force production during training may be limited and potential for adaptations reduced.

If It is shown that lumbar extensor torque affects hamstring torque, there could be implications for the screening and monitoring of athlete's injury risk. Eccentric peak torque is a common measure for associating or predicting injury risk (Green, Bourne and Pizzari 2018; van Dyk *et al.* 2016; Opar *et al.* 2015; Timmins *et al.* 2016). Athletes with weakened lumbar extensors might appear to be of greater risk of injury than the active force producing capacity of the hamstring would suggest. If the magnitude of effect is substantial, then pelvis restraints might be necessary to accurately estimate the force producing capacity of the hamstrings.

Therefore, the aim of this study was to compare hamstring torque before and after a lumbar fatiguing protocol equivalent to soccer compared to a control leg. It is hypothesised that lumbar extensor fatigue will reduce hamstring peak torque. Exploratory hypotheses will investigate if the reduction in peak torque increases with longer muscle lengths.

3.4.2. Method

3.4.2.1. Study design

Participants were required to attend two sessions separated by at least 72 hrs to remove residual fatigue. The first session provided a full familiarisation of the tests and the second was used for data collection. Using a matched pairs design, each limb per subject performed repeated measures of maximal eccentric and concentric strength testing of the knee flexors pre- and post- either a lumbar extensor fatigue protocol or a passive rest, using an isokinetic dynamometer (Norm, HUMAC). An isokinetic dynamometer was preferred to the Nordic curl or any other exercise as the velocity of contraction could be standardised and the trunk could be supported, so effects could only manifest from the erector spinae's actions on the pelvis. It also allowed for comparisons across the ROM unlike the Nordic curl.

Each limb was randomised to either the lumbar fatigue (experiment condition) or passive rest group (control condition) using the Excel 'RANDOM' function (version 2016; Microsoft, Reading), where odd numbers signified the control condition and even numbers signified the experimental condition. The dominant limb was assigned first, determined as the preferred kicking limb. Figure 15 provides an outline of the study procedure.

3.4.2.2. Participants

A convenience sample of 16 amateur footballers was obtained (age: 22 ± 5 years; mass: 73.5 ± 10.6 kg; stature: 175.9 ± 6.9 cm). Eleven participants competed between levels 4–15 (mode of 4) of the English national league and five participants competed in a university or college league. The sample consisted of five defenders, six midfielders, and five attackers. Participants were excluded if they were currently injured or experiencing any pain or soreness. Participants had to be male due to differences in the fatigue response to a given lumbar fatigue protocol (Stuart *et al.* 2018) and must compete at the amateur level of football (national league) as an outfield player.

Considering the novelty of this research and logistical considerations for recruitment, the sample size for this study was determined in order to detect effects accounting for no less than 25% of the reduction in hamstring torque after soccer. Prior research has reported a 46 N·m change in hamstring torque after simulated soccer (Small *et al.* 2010). A 25% reduction would equate to 11 N·m and, according to Small (2008), an effect size of 0.71 (intra-subject r = 0.94). To achieve an alpha of 0.025 (two hypotheses for concentric and eccentric modality) and a beta of 0.8, it was determined that 18 participants would be required for a paired samples t-test.

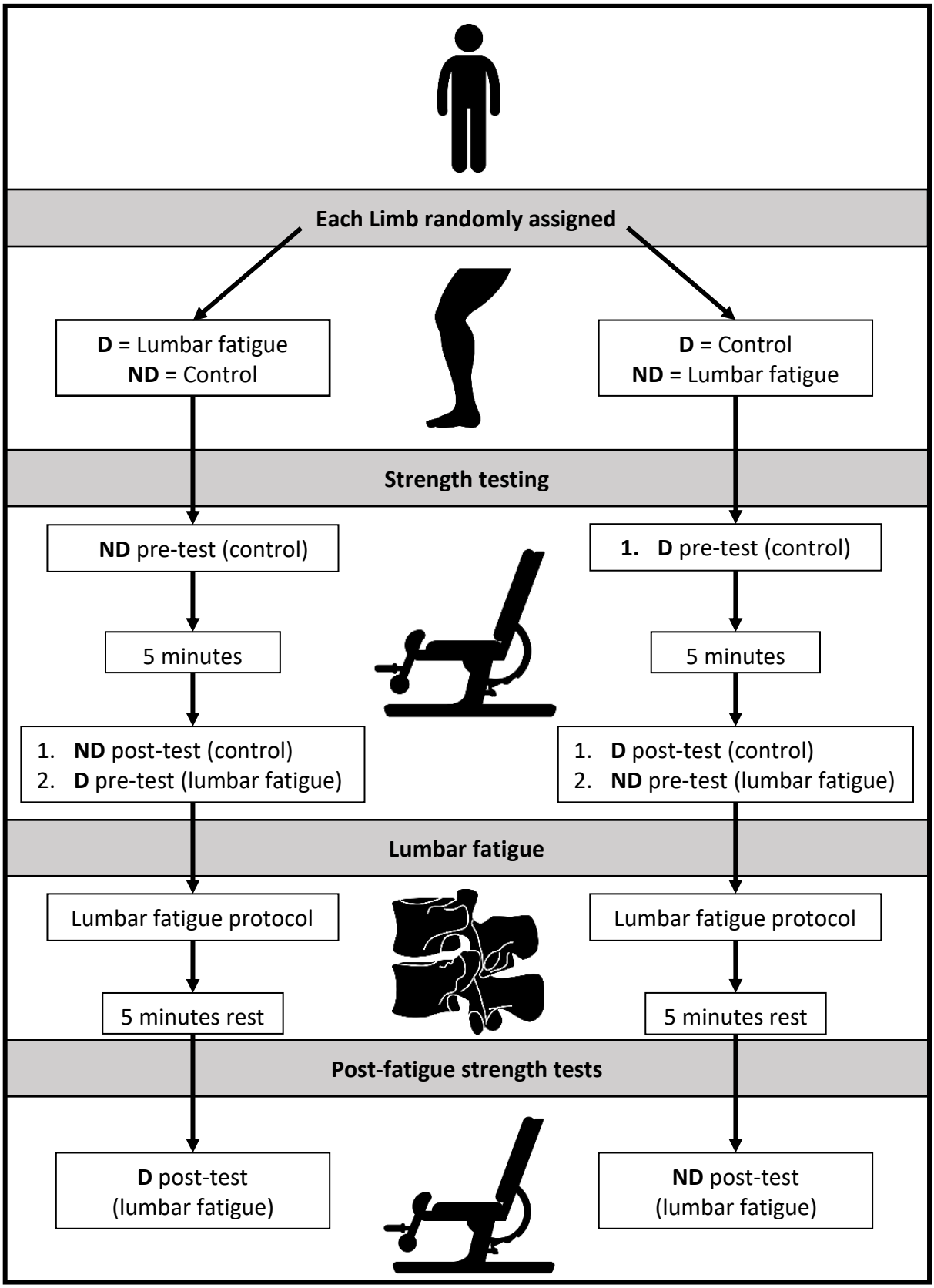

Figure 15: The isokinetic testing procedure and participant allocation. **Note:** D= Dominant; ND= Non-Dominant.

3.4.2.3. Procedure

The familiarisation session consisted of the baseline eccentric and concentric strength testing for each limb and performing the isolated lumbar extensor fatigue protocol.

3.4.2.4. Isokinetic testing

Initially, participants cycled (874 E, Monark, Sweden) between 68 and 72 W for a duration of five minutes (van Dyk, Witvrouw and Bahr 2018; Dauty, Menu and Fouasson-Chailloux 2018; Lee *et al.* 2018). During this period, participants were briefed verbally of the nature of the isokinetic test to facilitate familiarisation.

Whilst seated, the distance from the knee flexion-extension axis to the lateral malleoli was measured (201, Seca, UK) for standardised placement of the dynamometer lever arm. The lever arm was set to a length 5 cm less than the tibia segment, so the attached calf pad was just proximal of the lateral malleoli. Participants were seated on the dynamometer with the chair upright (90°) and the back translation adjusted so the popliteal fossa was not touching the seat edge. A series of restraints were attached for reliable outcomes (Otten, Whiteley and Mitchell 2013). These included a chest and waist belt, a strap across the thigh of the tested limb, and a contralateral limb stabiliser. The knee joint axis of the testing limb was aligned with the lever axis by adjusting the chair position in the sagittal plane and the dynamometer height, ensuring the tibia made contact with the centre of the calf pad at approximately 90° of knee flexion. Axis alignment was confirmed by extending and flexing the limb to check for calf pad movement. Finally, the limb was weighed in full extension for gravity corrected torque outputs. Dynamometer configuration was assumed to be symmetrical for each limb and so the input for the first limb was used for all further testing with the exception of gravity correction.

After dynamometer configuration, participants performed five practice repetitions consisting of two continuous concentric-eccentric repetitions at 50% of self-determined effort on each limb. This was followed by three maximal repetitions. Thus, each limb performed two familiarisation trials

before data collection when considering the full familiarisation and the five practice repetitions prior to testing. Whilst a single familiarisation session has good reliability (ICC: 0.68), it is improved with an additional session (ICC: 0.84; Nugent, Snodgrass and Callister 2015). After three minutes of passive recovery, five maximal repetitions were recorded for baseline measures (Dauty, Menu and Fouasson-Chailloux 2018; van Dyk *et al.* 2016). Concentric repetitions began in full extension (0°) and terminated at full flexion (90°) whereas eccentric contractions returned to the start position (90° – 0°). All repetitions were performed at a velocity of 60 deg·s^{-1} for reliability purposes (Nugent, Snodgrass and Callister 2015) and with standardised verbal feedback using the command 'pull' (Lee *et al.* 2018). After a further 5-minute rest, the control limb was re-assessed for changes in strength using the above procedure. Participants then proceeded to perform baseline measures for the opposite limb, which was assigned to the fatigue condition. The lumbar fatigue protocol immediately followed. After a 5-minute passive rest, hamstring torque was re-assessed.

3.4.2.5. ILEX fatigue

The fatigue protocol was identified in Study 2 and has shown to be a valid procedure for inducing lumbar extensor fatigue equivalent to that experienced after 90 minutes of simulated soccer. Briefly, the fatigue protocol used an isolated lumbar extensor device to restrain the pelvis and isolate movement to the lumbar spine only. Following eight dynamic repetitions and three isometric MVCs at flexion (72°), upright (36°) and extension (0°) as a warm up, isometric peak torque was established by performing an MVC in lumbar flexion (72°), as peak torque consistently occurs in flexion (Stuart *et al.* 2018; Fisher, Bruce-Low and Smith 2013). To confirm peak torque, torque was also measured at a more extended angle of 62°. Participants were offered further attempts if they felt the test was not maximal. To fatigue the lumbar extensors, participants performed five extension repetitions across a full ROM (72° – 0°; 0° = full extension) using 80% of peak torque, immediately followed by five minutes of passive rest. Further details of this protocol can be found in Study 2.

3.4.2.6. Data processing

Peak torque values for pre and post in each limb were obtained by taking the mean of the three repetitions with greatest peak torque. For each mode (concentric and eccentric) and condition (control or lumbar fatigue), the absolute peak torque value for both pre and post-tests was used to calculate change scores (Pre – Post). For both modalities, mean change scores and their respective SD for each condition (control or lumbar fatigue) are reported along with the net change (change in fatigue trial minus the change in the control trial). The torque produced at each 10° of the ROM (10°–80°) was also obtained and processed identically to peak torque to calculate the net torque change across the ROM.

3.4.2.7. Statistical analysis

For the concentric modality, the change in torque for the control group was not normally distributed (Shapiro-Wilk's: $p < 0.01$). The change in torque for the fatigue group and the net change in torque were both normally distributed. Therefore, descriptive statistics for the torque change in the control group used medians and interquartile ranges but means and SDs for the fatigue group and the net change. For the eccentric modality, change in torque data were normally distributed for both conditions (control and fatigue) along with the net change data (Shapiro-Wilk's $p > 0.05$). Therefore, data were described using means and SDs. These statistics were obtained using JASP (version 0.14.1). Exploratory analyses were performed by comparing descriptive statistics to test the notion that the reduction in torque was greater as muscle length increased (smaller knee flexion angle). The net torque change was not normally distributed at some angles, thus comparisons of the torque change at each angle were made using medians and interquartile ranges.

The effects of lumbar extensor fatigue on torque were assessed via a linear mixed model using R statistical software (R Core Team 2018; Kuznetsova, Brockhoff and Christensen 2017; Bates *et al.* 2015) where the change in torque for each condition was modelled using 'condition' and 'baseline torque' as fixed effects and the 'individual' as a random effect. The 'individual' random intercept accounts for the variability between subjects, which results from within-subject clustering across

conditions (limbs). The baseline torque fixed effect accounts for changes due to regression to the mean. Linear mixed-models are inherently more powerful than the paired t-test, so the sample estimate of 18 participants served as a conservative estimate. In addition, a hypothesis regarding either concentric or eccentric strength was not specified, but as both the hypotheses predict a one-sided effect, no error correction was necessary as the tail of a two-sided t-test with an alpha of 0.05 represents 2.5% of data. To assess if reductions in hamstring torque were greater as muscle lengths increased, the net change in torque at 10° intervals between 80°–10° was visually presented for both concentric and eccentric modalities.

3.4.3. Results

For the concentric modality, the control group torque increased by 4 N·m with an interquartile range of 20 N·m. In the ILEX fatigue condition, torque reduced by −10 ± 25 N·m. The net change in torque for the concentric modality was −19 ± 23 N·m. In the eccentric modality, the control group torque increased by 12 ± 19 N·m. In the ILEX fatigue condition, it reduced by −14 ± 22 N·m. The net change in torque was -26 N·m ± 27 N·m.

Results of the linear mixed model show hamstring peak torque was significantly reduced after lumbar extensor fatigue for both concentric and eccentric modalities compared to controls (F = 10.371, Numerator df = 1, Denominator df = 14.884, p = 0.006 and F = 21.296; Numerator df = 1; Denominator df = 13.76; p = 00004, respectively). Visual comparisons show the reduction in torque was greater as the knee became more extended for both concentric and eccentric modalities, though the effect was more consistent in concentric actions (figure 16). The net change in concentric torque at 80° was -3 (17) N·m and at 10° this had become -24 (59) N·m. For eccentric torque, the net reduction at 80° was -4 (31) N·m and at 10° torque reduced by -17 (67) N·m.

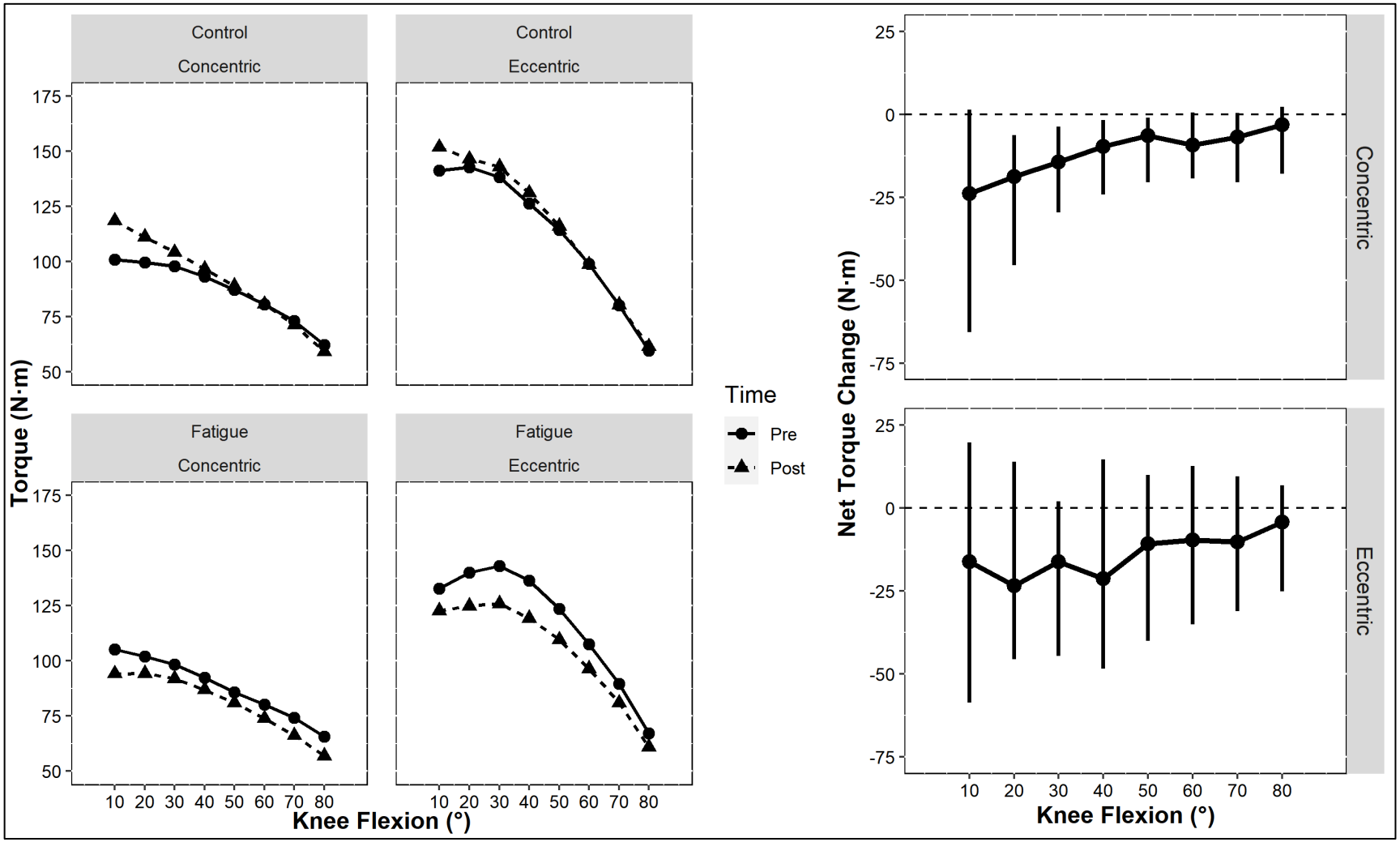

Figure 16: Isokinetic hamstring torque before and after ILEX fatigue. Left: Hamstring torque during eccentric and concentric modalities for the control (top row) and fatigued limbs (bottom row). For the control limbs, post trials represent torque after five minutes passive rest. Right: Median and interquartile range for the net change in the post trials for the fatigued limb compared to the control limb for concentric and eccentric modalities.

3.4.4. Discussion

The aim of this investigation was to measure peak torque in the hamstrings during maximal contractions before and after soccer equivalent ILEX fatigue. It was hypothesised that the induced lumbar extensor fatigue would reduce hamstring peak torque. The findings from this investigation support the hypothesis, with peak torque reducing by −10 ± 25 and −14 ± 22 N·m for concentric and eccentric contractions respectively. Furthermore, the net reduction in torque (change in fatigue condition minus the change in control condition) was −19 ± 23 N·m for the concentric modality and -26 N·m ± 27 N·m for the eccentric modality. This is the first study to demonstrate that a stabilising muscle during hamstring actions can affect torque measurements.

Halperin, Chapman and Behm (2015) acknowledged the potential for biomechanical fatigue through muscle stabiliser weakness, however, they also acknowledged that non-local fatigue could occur through neurological, psychological, and biochemical factors. Biochemical fatigue is the presence of metabolites that have been distributed to non-local muscles via the cardiovascular system. It seems unlikely hamstring metabolite fatigue exists after the five repetitions of lumbar extensions and five minutes rest, as the hamstrings show no change in twitch torque throughout a 90-minute soccer simulation (Marshall *et al.* 2014) and resistance training with an inter-set rest above two minutes is sufficient for strength gains (Grgic *et al.* 2018), suggesting intact muscle performance. Psychological fatigue (motivation) is also unlikely to explain the reductions in hamstring force as standardised verbal feedback was used with subjects confirming trials were maximal. These steps align with Gandevia's (2001) recommendations to ensure contractions are maximal efforts. Neurological fatigue occurs through group three and four afferents decreasing central drive in non-exercised muscles (Halperin, Chapman and Behm 2015) and may explain the hamstring fatigue in this study, but this seems unlikely. Place *et al.* (2004) showed no change in hand grip strength after five hours of running and the findings of Thomas *et al.* (2017) indicate reductions in voluntary activation are limited to the muscles performing work. Similarly, study 1 failed to identify a reduction in hand grip force after 90 minutes of soccer simulation. Indeed, a

recent meta-analysis found no evidence of non-local muscle fatigue (Behm *et al.* 2021). If neurological fatigue was responsible for the findings in this study, eccentric torque should reduce more than concentric torque as greater neural drive is required for eccentric actions (Barrué-Belou, Marque and Duclay 2018) but this was not observed.

The most convincing evidence of biomechanical fatigue is presented in figure 16, where reductions in torque increase as the knee extends. If any biochemical, psychological, or neurological factors were responsible, one would expect a reduction in torque across the entire ROM. This was not the case, and the reductions in torques at longer hamstring lengths are in alignment with the hypothesised posterior rotation of the pelvis as the lumbar extensors cannot oppose the pull of the hamstrings. Thus, biomechanical fatigue is the most plausible explanation for the reductions in hamstring torque observed in the study. This is in contrast with the notion that non-local muscle fatigue does not exist (Behm *et al.* 2021), and future research should investigate the possibility of biomechanical fatigue (as defined by Halperin, Chapman and Behm 2015) more broadly.

Previous investigations have shown pelvic compression belts increase average eccentric hamstring torque at long muscle lengths (25°–5° of knee flexion) by 0.1 N·m per kg of body mass (~7.12 N·m; p = 0.044; Arumugam *et al.* 2014b). Perhaps hamstring force increases with pelvic compression because it lowers erector spinae demands, and thus it has a greater capacity to stabilise the pelvis during knee flexion efforts. Indeed, multifidus activity is decreased whilst wearing a pelvic compression belt during gait (Arumugam *et al.* 2015) and unilateral standing (Arumugam *et al.* 2014a) in the same group of participants (although the reductions are small ~4% and ~1% of MVIC respectively; $p < 0.05$).

Lumbar extensor fatigue appears to explain between 22–46% of the reductions in hamstring torque after soccer (Marshall *et al.* 2014; Greig 2008; Small 2008). Marshall *et al.* (2014) found the SAFT90 protocol induces central fatigue in the hamstrings but not peripheral fatigue. Combined with the findings of this study, it would seem reductions in hamstring torque after soccer arise from a

combination of neurological and biomechanical fatigue. Currently there seems to be a wealth of research towards improving hamstring morphology, architecture, and strength but very little addressing the stabilisers for hamstring actions (Bourne *et al.* 208). This is an area that needs more work as the stabilisers could place a limit on the improvements gained from hamstring strengthening.

Eccentric torque is often used to predict injury risk or identify associations to injury (Green, Bourne and Pizzari 2018; van Dyk *et al.* 2016; Opar *et al.* 2015; Timmins *et al.* 2016). Yet if pelvis rotation occurs because of excessive or insufficient force from the stabilising muscles, it could lead to misleading results. The results of this study suggest an athlete with weak or fatigued lumbar extensors would appear to be at greater risk of injury than the force generating capacity of the hamstring would suggest. This might partly explain why a consistent association between eccentric hamstring torque and injury risk is not always found (Opar *et al.* 2021). It is commonplace to use thigh and chest restraints during isokinetic testing to prevent trunk and hip motion, but the pelvis is free to rotate. More precise estimates would be obtained from using a pelvic restraint during knee flexion testing.

Whilst this study implemented a control condition to mitigate any learning or fatiguing effects from the baseline hamstring strength assessment, it does not rule out hamstring fatigue that could have occurred during lumbar extensor fatiguing contractions, as the hamstrings attempt to posteriorly tilt the pelvis. Nonetheless, this seems unlikely to explain the findings observed here as reductions in hamstring torque increased as the knee became extended and with little fatigue at shorter lengths (figure 16), which would not be expected with local peripheral fatigue. Further hamstring activity is relatively small during ILEX actions (Udermann *et al.* 1999). It may be that the 5-minute rest period after lumbar extensor actions removes any peripheral fatigue that occurs in the hamstrings.

3.4.5. Conclusion

This study has found ILEX fatigue equivalent to what is experienced after soccer reduces hamstring peak torque and this effect is most notable at longer hamstring lengths. It is the first study to provide evidence of the interaction between the pelvis stabilising muscles and force production in the hamstrings. This has important implications for the testing of hamstring force when the pelvis rotation is not controlled, and for training programmes where lower lumbar extensor force might limit adaptations in the hamstrings. Research is needed to investigate whether strengthening the lumbar extensors facilitates adaptations in in the hamstrings. It is also important to investigate whether hamstring torque is reduced with lumbar extensor fatigue when the knee angular velocity is much greater, such as in running.

SECTION 4:

INTERPRETING THE FINDINGS

4.1. SUMMARY OF FINDINGS

Throughout this thesis a series of important steps have been taken to improve our understanding of hamstring strain injuries and their prevention. Core training is widespread in injury prevention programmes (Meurer, Silva and Baroni 2017; McCall, Dupont and Ekstrand 2016; Bahr, Thorborg and Ekstrand 2015) but this is surprising as there is little research suggesting it is of benefit. In fact, the ubiquity of core training suggests it is of some divine importance that has largely gone unquestioned. The first suggestions of the core's involvement in injury mechanisms dates back to Tucker in 1954, and it seems it was not until 50 years later that the first evidence emerged showing the promise of core strengthening (Sherry and Best 2004). Yet the work of Sherry and Best (2004) is far from conclusive. Since then, Schuermans *et al.* (2017a), Daly (2017), and Bonte *et al.* (2015) have identified differences in trunk and pelvis kinematics between injured and uninjured participants but the core muscles responsible, if any, remain unknown (Schuermans *et al.* 2017b), though Bonte *et al*. (2015) did observe reduced ES activity in injured subjects. Therefore, the purpose of this thesis was to answer the question "can core muscle weakness increase HSI risk in soccer players?".

The current literature indicates the ES is the most likely core muscle to increase HSI risk. This was based on expecting lumbar extensor fatigue to reduce lumbar extension during stance, increasing the trunk lean and subsequently rotating the pelvis anteriorly (Higashihara *et al.* 2015), and because of its apparent high activity during running (Saunders *et al.* 2005) indicating this muscles susceptible to weakness and fatigue from soccer. Although without empirical support (Bickham, Young and Blanch 2000), the trunk flexors also possess a mechanism to increase anterior pelvic tilt if weakened based on their anatomical connections to the pelvis.

The empirical findings of this study identified the following: Lumbar extension torque does not differ to recreationally strength trained individuals and powerlifters, emphasising that lumbar extension strength cannot be increased using typical resistance-based exercises. The lumbar

extensors are fatigued more than the trunk flexors during soccer, and the fatigue cannot be explained by non-local factors. Considering their high level of activity compared to the trunk flexors (Saunders *et al.* 2005), it was concluded the lumbar extensors were more likely to be weak than the trunk flexors. The magnitude of lumbar extensor fatigue from soccer can be replicated using five repetitions of lumbar extensions with a weight equivalent to 80% of peak torque, allowing for studies to use this protocol for causal inference. Lumbar extensor fatigue after soccer increases anterior pelvic tilt during portions of running, and reduces eccentric and concentric hamstring torque, which was more notable as the muscle length increased, indicative of biomechanical fatigue.

The results of the empirical studies provide evidence to show that lumbar extensor fatigue from soccer does indeed increase the risk of HSIs according to the model of injury proposed in figure 3. The increase in anterior pelvic tilt during the pre-swing phase (when the contralateral limb is in terminal swing) with no change in the knee angle, indicates increased hamstring lengthening and thus hamstring stress. While the increase in anterior pelvic tilt is relatively small (10% of the increased anterior tilt in injured subjects; Schuermans, *et al.* 2017a), it would be incorrect to conclude lumbar extensor fatigue is not a cause of HSIs according to the proposed model of injury. As result, the original model conceived from the literature is now updated to include the demonstrated link between the lumbar extensors and anterior rotation of the pelvis during running (figure 17). Though this thesis has demonstrated lumbar extensor weakness can increase anterior pelvic tilt, the magnitude of the difference raises some concerns for the wider adoption of core training.

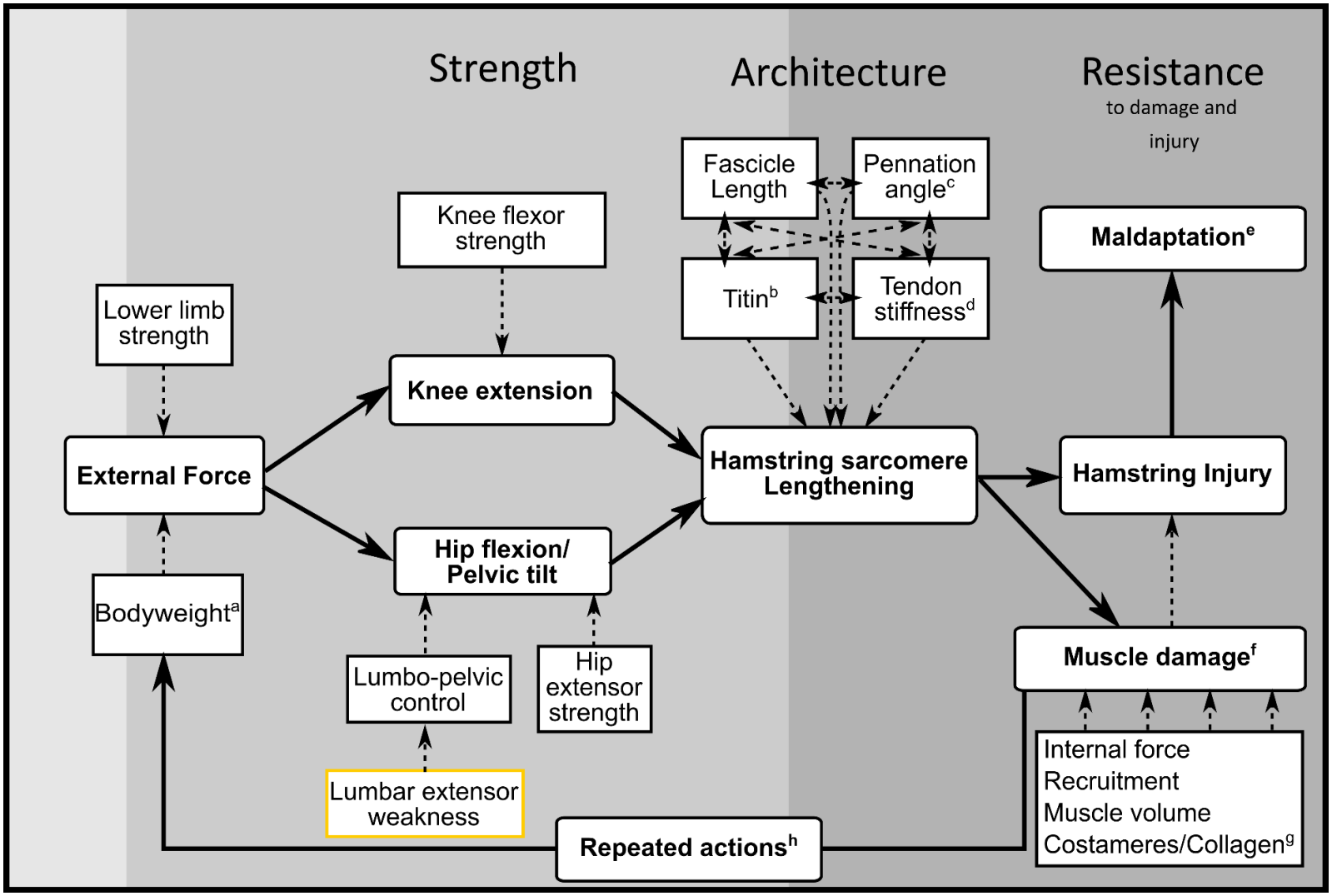

Figure 17: The updated causal model of HSIs. **Note:** Dotted boxes show the new information in the model. †examples of extracellular matrix variables. Solid lines show the path between cause and effect. Dashed lines represent moderating variables.

The lumbar extensors were empirically investigated because they appeared more likely to be weak than the trunk flexors in soccer players. Compared to the trunk flexors, they display a higher level of activity during running (Saunders *et al.* 2005), produce greater torque whilst running (Sado *et al.* 2019) relative to their force generating capacity (Fransson *et al.* 2018), and experience greater fatigue from soccer (study 1 and Fransson *et al.* 2018). Thus, the trunk flexor fatigue from soccer is expected to have an even smaller effect on anterior pelvic tilt, if an effect occurs at all. There is evidence showing no relationship between the ability to resist anterior pelvic tilt at baseline (without fatigue) and anterior pelvic tilt when running (Bickham, Young and Blanch 2000). So, given this and the small magnitude of increase in anterior pelvic tilt with lumbar extensor fatigue, it is reasonable to propose that anterior pelvic tilt would not change after soccer because of trunk flexor fatigue. Though of course, one needs to consider not just the susceptibility to weakness but the mechanism of the increased anterior pelvic tilt with trunk flexor fatigue too. Whilst trunk flexors weakness appears unlikely, it does possess a mechanism to increase anterior pelvic tilt if it were to become weak based on its anatomical attachments to the pelvis (table 4). Yet this seems unlikely to occur as resisting pelvic tilt during running would reduce stride length and sprint performance (Franz et al. 2009)

Taken together, the findings of this thesis raise important questions for the utility of core training to prevent HSIs. Particularly as core training programmes overlook the posterior core muscles. The FIFA11+ programme is designed to be comprehensive and address 'core stabilization' (Sadigursky et al. 2017) yet no single exercise targets the posterior core muscles whereas the anterior core muscles are prescribed two exercises (30% of all strengthening tasks). The intervention prescribed by Sherry and Best (2004) in attempt to reduce HSIs did not include any posterior core strengthening, and the core exercises included made reference to using the 'abdominal and hip' muscles. If core training is going to be used for the purpose of preventing HSIs, there is a stronger argument for strengthening the posterior core muscles than the anterior core muscles, and this

contrasts with what is current practice. Though training the posterior core is not easily done without pelvic restraints (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013).

Considering the barriers to lumbar extensor strengthening and the small changes observed in anterior pelvic tilt with lumbar extensor fatigue after soccer, and the fact the rectus abdominis seems unlikely to become sufficiently weak to alter anterior pelvic tilt, it seems reasonable to conclude that core strengthening for HSI prevention should not be done at the expense of proven injury reduction practices, such as eccentric training (Petersen *et al.* 2011) and high-speed running (Duhig *et al.* 2016). This is concerning as McCall *et al.* (2014) found 100% of 44 soccer clubs used core strengthening yet only 79.5% of clubs used eccentric training. Though this thesis disagrees with the notion that core strengthening is not necessary at all (Lederman 2009), as anterior pelvic tilt was increased with lumbar extensor fatigue. At the individual level, there may be merit to including lumbar extension strengthening if a noticeable forward lean is observed whilst running or if athletes suffer from chronic HSIs despite already utilising common preventative measures (i.e. Nordic curls).

Although strengthening the posterior core is more justified than the anterior core, this is based on the evidence of this thesis and the running kinematics of the participants who took part. It is expected that these findings can be generalized to other amateur soccer players and likely other sports too, but recent evidence has found the angle of anterior pelvic tilt during waking can be altered with 'corrective exercises' and mobility training (Mendiguchia *et al.* 2020). It is unclear if this would transfer to soccer match play, as walking was assessed rather than sprinting, and although the study was single blinded, there were numerous exercises that encouraged posteriorly tilting the pelvis, which may have altered the behavior of participants in the post test trials. Nonetheless, an intervention like this may increase activity in the anterior core muscles and justify the inclusion of strengthening these muscles. More research is needed to understand if trunk flexor weakness is likely to occur when adopting a reduced anterior pelvic tilt during running and thus if strengthening these muscles is justified for these conditions.

This thesis also found eccentric and concentric hamstring torque was reduced with lumbar extensor fatigue. This observation has not been added to the conceptual mechanism of HSIs (figure 17) because this phenomenon was not observed during running where angular velocities at the knee are much greater. Indeed, it is not expected to be observed during running as hamstring force, and thus the erector spinae force will be reduced with the much greater angular velocities. On the other hand, strengthening the lumbar extensors may be beneficial for improving hamstring strength and facilitating adaptations by maintaining the pelvis angle during forceful hamstring actions. Failure to maintain the pelvis angle would result in posterior rotation of the pelvis and a loss of passive tension. The evidence from this thesis suggests exercises performed after soccer would produce lower hamstring forces, partly because of lumbar extensor fatigue. This may limit the adaptations from exercise. For example, Lovell *et al.* (2018) noted no increase in fascicle lengths when Nordic curls are performed after soccer. More work is needed to assess if lumbar strengthening is necessary as the hamstrings become stronger from chronic training to maintain the pelvis angle and thus hamstring forces. In addition, this thesis has shown evidence of biomechanical fatigue in the hamstrings due to weakness in a more proximal muscle (i.e. lumbar extensors). Ultimately this shows pelvis rotation can influence measures of hamstring force and could have an effect on the validity of these assessments. Practitioners should consider this when selecting the modality of hamstring strength assessments and their parameters. It may be more valid to consider tests that measure hamstring strength at a shorter ROM, such as the Nordic curl, rather than at long lengths where the pelvis may have an influence on the findings by manipulating the passive tension (figure 16).

Further to the empirical findings, this thesis has proposed stress as the principal cause of muscle injuries. To date, work towards identifying the hamstring muscle most at risk of injury during running has not included stress, and instead measured the strain or forces (Chumanov, Heiderscheit and Thelen 2011; Thelen *et al.* 2005a; Nagano *et al.* 2014). By measuring stress, it is hoped that future work will be able to more clearly identify the hamstring muscle most at risk and its causes.

The thesis is also the first to develop a causal model of the HSI mechanism. Many studies towards HSIs investigate the effects of interventions on surrogate markers that are lacking supporting evidence (Alonso-Fernandez, Docampo-Blanco and Martinez-Fernandez 2017; Chen *et al.* 2011; Cameron *et al.* 2007). This framework makes clear the factors that should be examined and variables that could confound any conclusions and can be updated in light of new evidence.

Although this thesis has made important steps to understanding the role of the core muscles in HSIs, there are inevitable limitations. Whilst steps were taken to isolate the lumbar extensor involvement in the fatiguing protocol, the isometric action of the hip extensors could not be eliminated. Though it seems unlikely that hip extensor fatigue occurred; there was an angle-specific reduction in hamstring torque after lumbar fatigue, which is consistent with biomechanical fatigue rather than local fatigue. There was also no change in the hip angle during stance or during hip flexion in swing when the hip extensor torques are greatest (Schache *et al.* 2011), and a small number of contractions (five repetitions) were used in the fatiguing protocol along with a low level of activity (Udermann *et al.* 1999) and five minutes passive rest. Unfortunately, it is not possible to exclude the hip extensors involvement entirely. Another limitation was that only amateur soccer players were used. It could be argued that amateur players are weaker than their professional counterparts and therefore more susceptible to the findings here. Yet, this in fact strengthens the findings. As the lumbar extensors cannot be strengthened during typical exercise (Hammond *et al.* 2019; Fisher, Bruce-Low and Smith 2013), the greater strength in professional athletes would suggest the already deconditioned lumbar extensors would have to oppose even greater forces during match play and fatigue even further. Therefore, the increase in anterior rotation of the pelvis may be even greater than observed in this thesis.

4.2. CONCLUSION

The key finding from this thesis is that soccer-equivalent lumbar extensor fatigue increases the risk of hamstring strain injuries by anteriorly rotating the pelvis during running, in turn lengthening the

hamstrings. It has also been shown that the lumbar extensor fatigue from soccer can reduce hamstring torque output, potentially limiting adaptations from hamstring exercises. The increase in anterior rotation of the pelvis with soccer equivalent lumbar extensor fatigue appears small and lumbar extensor strengthening should not be prioritised over more established prevention techniques such as eccentric hamstring strengthening. The small change in anterior rotation of the pelvis with soccer equivalent lumbar extensor fatigue raises concerns of the utility of core muscle strengthening for HSI prevention, particularly because of its global adoption and preference to eccentric muscle strengthening.

SECTION 5:

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SECTION 6:

APPENDICES

6.1. ETHICAL APPROVAL FOR STUDY 1 (PART 1)

OUTLINE OF PROPOSED RESEARCH TO BE SUBMITTED FOR ETHICAL APPROVAL (FORM HESS01)

PLEASE NOTE: Students will need to discuss this form with their dissertation tutor for each project undertaken. Before completion the applicant is advised to consult the Health Exercise and Sport Science Ethical Policy available on the mycourse under the unit code of LEI422. In addition, the applicant should also read and understand the British Association of Sport and Exercise Sciences (BASES) Code of Conduct and if the dissertation falls within the auspices of psychology, then you are advised to ensure you have read and understood the Ethical Principles for Conducting Research with Human Participants published by the British Psychological Society.

This form must be completed by Staff, Postgraduates and Undergraduates before potential participants are approached to take part in any research. Please complete all questions. A copy of your proposal/abstract will be attached to this HESS01 form.

Project Name:	A study to measure the magnitude of lumbar extensor fatigue expereinced in competitive footballers after simulating a football match.
Area to be studied:	Physiology Please specify...
Proposed Start Date:	11/06/2017
Proposed Duration:	11/08/2017
Principal Investigator:	Craig Perrin
Contact No:	07791201502
Level:	MPhil/PhD
Year of study:	1
Supervisor:	Dr James Steele
Other Investigators:	Dr Brian Wink, Dave Smith

Question	Yes	No
Q1a. Does your study involve the use of human participants?	◉	○
Q1b. Minors (under 18 years of age) or vulnerable adults?	○	◉
Q1c. Overt observation techniques, such as notational analysis?	○	◉
Q1d. Questionnaires or semi-structured interviewing?	○	◉
Q1e. The discussion of sensitive topics?	○	◉
Q1f. Covert observation or deceptive procedures?	○	◉
Q1g. Sub-maximal exercise testing (i.e. less than 85% heart rate) This may also involve testing flexibility testing, massage techniques?	◉	○
Q1h. Physiological testing that is maximal in nature and greater than that experienced in everyday life? This might include activities such as one repetition maximum testing, time-trialling or maximal exercise testing (VO2 max test).	◉	○
Q1i. High risk psychological or physiological distress or harm to participants that exceeds normal life. This might include activities such as:	○	◉

- Paramedic or medic in attendance during a resting ECG and throughout experimental testing
- Clinical trials
- Research on abnormal or clinical psychology
- In addition, participants are children under 5, pregnant women or vulnerable adults.

Q2. Describe the research problem / issue under investigation.

Hart et al. (2009) found fatiguing the paraspinal muscles increased trunk flexion during steady state jogging. However, the causation of these findings cannot be solely attributed to paraspinal fatigue as the hip extensors likely contribute to trunk extension when the pelvis can rotate freely (Fisher, Bruce-Low and Smith 2013). Similarly, Increased hip flexion is also present during quiet standing after fatiguing the lumbar extensors (Madigan, Davidson and Nussbaum 2006), a condition which reduces the numerous variables that could influence the forward lean in comparison to running. Trunk flexion appears to be a consistent symptom of lumbar fatiguing protocols though none specifically isolates this musculature.
Furthermore, it is expected that changes in kinematics at the spine and hip (e.g. trunk flexion) would consequently alter kinematics in the lower limbs. Higashihara et al. (2015) noted that increased forward lean during sprinting is accompanied by anterior pelvic rotation and increased flexion at the knee. From the available literature, it would seem the lumbar extensors might be partly responsible for the changes in kinematics induced from fatigue. A common theme throughout aforementioned research is the use of a fatiguing protocol that lacks external validity to very few sporting scenarios. Indeed whilst it is likely fatigue of the lumbar extensors may cause trunk flexion, it is not known whether this degree of fatigue can occur under sport specific circumstances. Furthermore, the fatiguing protocols used throughout the research fail to isolate the lumbar extensors and so causation cannot be inferred definitively.

Q3. What is the rationale that underpins the study (i.e. why is the study worth conducting?).

Typically, the lumbar extensor musculature is of great interest in low back pain research but its potential to impair performance seems overlooked. Indeed, the cross-sectional area of the erector spinae and quadratus lumborum are significantly correlated to sprint performance from 0-20m and combined they explain 50% of the velocities achieved over 20m (R2= 0.504; Kubo et al. 2011), yet the reasons for such findings is unclear. Considering sprinting is a key movement in many sporting events, any possible impairments should be investigated to minimise potential risks to performance.

Q4. What is the aim of the study (i.e. what are you looking to investigate)?.

The aim of this study is to understand the magnitude of fatigue that occurs within the lumbar extensors after simulating a

competitive football match by measuring the loss in torque. This will allow future studies to assess whether this loss in torque can alter sprint kinematics.

Q5. Which methodological approach are you looking to adopt?. Quantitative

Q6. Which research design are you looking to adopt?. Experimental

Please specify...

Q7. Provide details of the questionnaires, protocols, techniques and procedures to be used during your data collection (e.g. CASI, Wingate test, capillary blood sampling, semi-structured interviewing).

The MedX Lumbar extension measures lumbar strength isometrically across the individual's full range of motion (normally from 0°to 72° at 12° intervals) and will be executed according to the manufacturer's guidelines.

The simulation of the football match (SAFT90 protocol) shall last 90 minutes with a 15-minute passive rest at the half way point. The course is 20 meters in length and involves a variety of movements such as sprinting, sidestepping and walking. The instructions are standardised and pre-recorded on an audio file. This protocol is designed to replicate the physiological and mechanical demands of a competitive football match.

Q8. How do you intend to analyse your data (e.g. content analysis, one-way repeated measures ANOVA)? Please provide specific details.

Differences in lumbar torque pre and post football simulation shall be measured using either a paired samples t-test or a Willcoxon signed rank test dependent on the outcome of the Kolmogorov–Smirnov test with significance set at $P < 0.05$. 95% confidence intervals shall also be calculated for the loss in torque.

Briefly describe the sample demographic (e.g. age, gender etc.) and the sample method (e.g. convenience, purposive etc.).

Participants should be purposively sampled to be male, adult (over 18yrs), competitive footballers. Participants must not have any malignancy or underlying disease, disc herniation, osteoporosis, neurologic or sciatic nerve root compression, previous vertebral fractures, major structural abnormality of the spine, problems passing fluid or solids and inflammatory arthritis. Furthermore, participants must not currently be suffering from any injury or be undergoing rehabilitation for any injuries.

Q10. Methods of recruiting participants (e.g. poster, social media, team briefing etc.).

Individuals who meet requirements of the study will be given the opportunity to take part via email where no external pressure to persuade individuals to take part will be present. All participants will receive a full description of the study so participants are clear on what to expect if they do wish to take part. Local football clubs are expected to be the main source of recruitment.

Q11. Where will the study take place (e.g. University ? Physiology laboratory, a named school, a named hospital etc.)?.

Southampton Solent University laboratory.

Q12. Give an estimate of the amount of time you will require of each participant in the study/project. Please break this down into potential sub-headings like briefing, testing / training, debrief etc.

Session 1 (Familiarisation): 1 hour

10 minutes briefing/medical questionnaire
25 minutes for football protocol familiarisation
20 minutes for Lumbar extensor strength familiarisation
5 minute debrief

Session 2 (baseline testing): 0.5 hour

10 minute briefing/medical questionnaire
15 minutes lumbar extensor baseline strength testing
5 minute debrief

Session 3 (fatigue response test): 2.5 hours

10 minute briefing/medical questionnaire
115 minutes football simulation protocol
15 minutes for lumbar extensor strength testing
5 minutes debrief

Q13. How do you plan to handle the requirement of participant confidentiality? (e.g. password protected laptop or file)

All data shall be kept on a password protected laptop and will only be available to the relevant researchers. All participants will be assigned a code to replace names to ensure anonymity.

Question	Yes	No
Q14. Does your study have the potential for "upsetting" participants (e.g. affective manipulation or asking them to divulge information on a traumatic life experience) and/or for identifying distressed or disturbed individuals?	○	◉
Q15. Do you intend to pay participants for their participation?	○	◉
Q16. Will you be using any form of deception?	○	◉
Q17. Will the study require the cooperation of a gatekeeper for initial access to the groups or individuals to be recruited? (e.g. school pupils, residents of nursing homes)	○	◉
Q18. Will it be necessary for participants to take part in the study without their knowledge and consent at the time? (e.g. covert observation of people in public places)	○	◉
Q19. Will the study involve discussion of sensitive topics (e.g. sexual activity or drug use)?	○	◉
Q20. Are drugs placebos or other substances (e.g. food substances, vitamins) to be administered to the study participants or will the study involve invasive, intrusive or potentially harmful procedures?	○	◉
Q21. Will blood or tissue samples be obtained from participants?	○	◉

Q22. Details of study

Please specify below.

The research will follow a repeated measures design with all participants completing three sessions. The first will form a familiarisation session using the ILEX device for reliability purposes (r=0.98; Graves et al. 1990), and to complete 15 minutes of the football simulation protocol (SAFT90;

Q22a. Physiological

If 'Yes' please check it falls into the categories ranked as safe in Appendix D of the HESS Ethics Document [] and list it below.

2o, 3i

Q22bi. Psychological

Q22bii. Other controversial or potentially risky manipulations

Q22biii. Do the hypotheses of your study involve the induction of negative effects upon the participants?(e.g. learned helplessness)

Q22c. In the case of questionnaire formats, will the study involve any questions, which may be upsetting?

Q22d. Nutrition

Q22e. Biomechanics

Q23. Is pain or mild discomfort likely to result from the study?

If 'Yes' please explain fully below.

Maximal lumbar extension strength testing will likely result in some muscular discomfort due to the maximal nature of the testing. Although this will be painless and subside shortly after testing. Likewise, the simulation of a football match may well result in some muscular discomfort.

Q24. Is there any risk to participants (physical and/or psychological) greater than that normally experienced in normal life?

If 'Yes' please explain fully what the risks are, how you plan to mitigate these, and justify their necessity below.

Gaining maximal lumbar extension strength will require a participant to generate more force than usual which may, although unlikely, result in muscle soreness in the following days. However, during the debrief clients will be given advice to reduce the chance of such symptoms occurring.

Q25 During your data collection will supervision or assistance be required? (e.g., for experiments in the physiology laboratory)

Q26. Will informed consent be obtained? (Unless there are very good reasons for not obtaining informed consent, the programme research guidelines state that you must use informed consent procedures)

Please insert your consent form below.

Informed Consent

Name of experiment: Lumbar extensor torque loss experienced in competitive footballers after the simulation of a football match.

1. I can confirm that the full details of the experiments/investigations have been explained to me. I am clear about what will be involved and I am aware of the purpose, the potential benefits and the potential risks. I can also confirm that I have had the opportunity to ask questions that I have about the experiment/investigation procedure.

2. I recognise that I have the right to withdraw my involvement at any time during the testing procedure.

3. Any data collected and stored on a computer will remain anonymous, however I understand that complete anonymity cannot be safe guarded due to the public nature of laboratory sessions.

4. I have completed a health questionnaire and agree to take part in this study.

Name of Participant Participants Signature Date
....

Declaration by the Academic Investigator

I can confirm that I have provided detailed information about the procedure which the above participant has consented to.

Name of Staff Staff Signature Date
....

Q27. Will a medical questionnaire need to be administered? (e.g. Physical Activity Readiness Questionnaire (PAR-Q form))

Q28. Will a pre-study questionnaire need to be administered? (e.g. International Physical Activity Questionnaire (IPAQ) - short version)

Q29. Does your project involve using children as your participant population?

Q30. Does your project use special participants (e.g., physically impaired / mentally impaired) as your participant population?

Q31. Is parental/guardian consent required for your project?

Q32. If undertaking questionnaire based research are you aware that you are required (i.e., this is not optional!) to have an ID card (your campus card will suffice) on show at all times and if collecting your data off campus you will be required to work in pairs (to maintain a safe environment) as well as informing one of your peers of your leaving time, location of destination and expected return time. By ticking yes you are agreeing to undertake this task.

Q33. Are frequent and repeated checks on your subjects required during the experiment?

Q34. Is a first aider required to be available during your data collection?

Q35. If your project requires you to have substantial unsupervised access to children under the age of 18, police screening needs to be carried out. This requires a Criminal Records Bureau Disclosure Form to be completed (see Appendix P for details). Does police screening need to be carried out?

Q36. Is a letter of competence required?

The following will be completed prior to data collection (please select as appropriate):

☑ Informed consent

☑ Physical Activity Readiness Questionnaire

Project category

Based on the questions on this form the following category is applicable (please answer question 1)

Participant Information Sheet (PIS)

Please copy and paste a copy of your PIS below.

Study title: A study to measure the magnitude of lumbar extensor fatigue expereinced in competitive footballers after simulating a football match.

Invitation:
We would like to take this opportunity to ask if you will consider taking part in our research into low back muscle fatigue. In order to help you make your decision we would like to inform you what this research will be measuring and the role you could potentially have in this study. The following text should take no more than 10 minutes of your time and if you have any questions, or if anything is not clear, please feel free to ask and we will be happy to answer them for you.

What is the purpose of the study?
The purpose of this study is to gain a deeper understanding of the role of the lumbar extensor muscles during football. In particular, we wish to examine if fatigue of this musculature can alter the way we sprint and its subsequent performance and injury risks. This study will be undertaken by Craig Perrin who is a Ph.D. student at Southampton Solent University, under the guidance of Dr. James Steele.

What will the study involve?
The study will require you to attend three sessions at Southampton Solent University sport science laboratories; The first session will take no more than 1 hour and is used to familiarise yourselves with the protocol. The second and third session will take approximately 30 minutes and 2.5 hours respectively and must be undertaken a minimum of 72 hours apart. All sessions will begin with a medical questionnaire and a blood pressure reading to ensure your safety when taking part in this study. I would like to add you DO NOT have to answer any of the questions if you do not wish to, however, they are only there to ensure your safety and may prevent you from taking part in the study. Each testing session will conclude with a debrief for you to ask any questions.

Testing day 1:
The first session shall consist of a familiarization test with the MedX lumbar extension strength test and the procedure for simulating a competitive football match. In order to isolate the low back musculature and prevent additional muscles assisting with the lumbar extensor strength test, the body, specifically the pelvis, must be properly restrained. A thigh and femur restraint (see Figure 1) prevents any upward movement of the knees, thighs, and pelvis whilst a footpad gently pushes the femur (leg bone) into the pelvis and preventing any muscles rotating the pelvis. Once properly restrained, we will assess the range of motion of the lumbar spine, normally from 0° to 72°, although this is not always the case. You will only be tested on the range of motion you can achieve. We will then move on to the dynamic warm-up consisting of seven repetitions of flexion and extension of the lumbar spine. Following this, a practice test, extending the lumbar spine isometrically (static) against a pad using only 50% effort shall be completed before moving on to the final test measuring maximal lumbar strength. This measurement is obtained similarly to the practice but this time across a full range of motion (0-72°) and using 100% effort.

The football simulation protocol is designed to replicate the mechanical and physiological demands of a competitive football match. The course is 20 meters in length and involves a variety of movements such as sprinting, sidestepping and walking (Figure 2). Verbal instructions using pre-recorded audio will instruct you with the movement (E.g. running or sidestepping) and the pace at which it should be performed (E.g. Walking or sprinting). The entire protocol will last 90 minutes with passive rest for 15 minutes at the half way point, replicating a football match. However, for the first session, you will only complete 15 minutes to familiarise yourselves with the procedure.

Testing day 2:
The second session will be at least 72 hours apart and will allow us to obtain a baseline measurement of maximal lumbar extensor strength. The protocol for lumbar extensor strength will be identical to the one used previously but will be completed much quicker as measurements for restraints will already be known. The football simulation protocol is not completed for this test

Testing day 3:
For the third and final session, the football simulation must be completed for the full 90 minutes immediately followed by a maximal lumbar extensor strength test. This will allow us to measure how much fatigue occurs in the lumbar extensors due to the completion of a football match. It is important to note another 72 hours must pass from the second session before completing this test to ensure no fatigue remains in the lumbar extensors from baseline testing.

Expenses and payments:
There are no expenses to take part in this study however, it will require you to travel to the University, which may require you to pay for transport.

Why have I been invited? ❑
For this research, we require competitive footballers who are free from injury and do not meet the exclusion criteria (see below). You have been invited because you meet these requirements.
Exclusion criteria:
If you suffer from any of the following conditions then, unfortunately, you will not be able to participate in the study for your own safety. If you are unsure of any of the following conditions then please feel free to ask and we can advise you on such matters; you will also complete a questionnaire regarding your ability to take part in this study, as your safety is paramount. The exclusion criteria include: no malignancy or underlying disease, disc herniation, osteoporosis, neurologic or sciatic nerve root compression, previous vertebral fractures, major structural abnormality of the spine, problems passing fluid or solids and inflammatory arthritis. Furthermore, you must not currently be suffering from any injury or be undergoing rehabilitation for any injury as this may influence the outcome of the study.

Do I have to take part?
It is up to you to decide whether to take part. If you do decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. Furthermore, you are free to withdraw at any time and without giving a reason. If you decide to withdraw we would like to stress this will not influence your treatment and you shall still receive a full debrief and ask any questions if you wish but the data we have collected up to your withdrawal may still be included in the study.

What are the possible disadvantages of taking part?
It is possible, although unlikely, you may feel stiff and sore, particularly within the low back, in the following 2-3 days of the study, however, this is likely to subside after a few days and should not interfere with your day-to-day life and is completely normal. This is because the muscles of your lower back are invariably not used to working by themselves. The researchers will also be able to give you advice to prevent such symptoms post testing.

What are the possible benefits of taking part?
There may be limited personal benefits to you in taking part in this study. However, we hope this study will help us to get a step closer to understanding the role of the low back musculature during sport and its potential role to hinder performance and increase injury risk. Furthermore, you will also be informed of your low back strength.

What happens when the research study stops?
Once the research stops, the data will be analysed appropriately. If you have any questions on the outcome of this research please feel free to contact us at any time and we will be pleased to inform you.
What if there is a problem?
Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed by a researcher instantly. If you remain unhappy and wish to complain formally, you can do this via Scott Burnet, Chair of the HESS Ethics Committee (ethics.hess@solent.ac.uk).

Will my taking part in the study be kept confidential? ❑
Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. The data gained from this study will be kept on a secured, password-protected laptop and data will only be shared with the relevant researchers. All names will be replaced with codes and so participants will remain anonymous.

What if new information becomes available?
Occasionally, new information becomes available on the topic under examination. If this happens, your researcher will discuss with you if you wish to carry on or not. If you do not wish to continue, your researcher will arrange for you to no longer take part, however, if you decide to continue with the study we may ask you to sign an agreement, obtaining your consent to continue.

Who has reviewed the study?
This study has been examined by the HESS ethics committee and has been reviewed and passed.

Contact details:
If you have any questions regarding the study whatsoever please feel free to contact Craig at Craig.Perrin@solent.ac.uk

Thank you.

Insurance Questionnaire (TIQ1)

Sponsor Name:	Solent University
Number of Participants:	20

Question	Yes	No
Q1. Is your research going to be based on Questionnaires?	○	◉
Q2. Is your research going to be based on Venepuncture?	○	◉
Q3. Is your research going to be based on measurements of physiological processes?	◉	○
Q4. Is your research going to be based on collections of body secretions by non evasive methods?	○	◉
Q5. Is your research going to be based on the administration by mouth of foods or nutrients or variation of diet other than the administration of drugs or other food suppliments or physiological activity (this is outside the Research definition)?	○	◉
Q6. Is the research to be held outside of the UK?	○	◉
If 'No' Please specify below. Southampton Solent University		
Q7. Who is involved in the research? Student, Supervisors and potential participants	○	◉
If medical practitioners are involved will they be covered by the MDU (Medical Defence Union) or any other organisation?		
Q8. Does the Research involve use of drugs or surgery?	○	◉
Q9. Are any of the research subjects (after enquiry) known to be pregnant?	○	◉
Q10. Are any of the research subjects (after enquiry) under 5 years of age?	○	◉
Q11. Does the Research involve genetic engineering?	○	◉
Q12. Will the Research use a pharmaceutical product designed or manufactured by the University?	○	◉
Q13. Will the sponsor pay for additional Insurance costs if required? (If ?No? please note that the school will be responsible for additional insurance costs)	○	◉
Q14. Is the purpose of the Research Investigating or participating in the methods of contraception?	○	◉
Q15. Is the purpose of the Research Assisting with or altering the process of contraception?	○	◉
Q16. If other organisations are involved in the Research, is SSU the lead organisation for the Research Project?	○	◉
Q17. Is the purpose of the Research Assisting with or altering the process of conception?	○	◉

If any of the answers to questions 4-10 are 'Yes' please provide full details below.

Note: Insurance cover against litigation
Projects sponsored by the University (almost all projects and all undergraduate studies) must be registered with the University's Insurance Department. This is done via Appendix C (see page 18). A project without a sponsor may well have no insurance cover if things go wrong. It is the researcher's responsibility to return the completed form to the programme administrator.

I/we, the investigator(s), confirm that:

☐ The information contained in this checklist is correct.
☐ I/we have assessed the ethical considerations in relation to the project in line with the HESS Ethics Committee.
☐ I/we understand that the ethical considerations of the project will need to be re-assessed if there are any changes to it.
☐ I/we will endeavour to preserve the reputation of the University and protect the health and safety of all those involved when conducting this research/enterprise project.

Signed by (ALL) the investigator(s): Date:

I confirm that, as supervisor:

- I have discussed the ethical considerations in relation to the project with the investigator(s) involved.
- I have read and agreed the information in this checklist.
- I will monitor progress of the project.

Signed: Print Name: Date:

HESS Ethics Committee Decision – PASSED With Conditions

Your ethics submission has been considered by the HESS Ethics Committee and the Committee has found there to be no major outstanding ethical issues. However, there were a number of important conditions which you must meet and are set out below. The Committee expects you to liaise with your Dissertation Supervisor to discuss these conditions and to ensure they are addressed prior to your data collection. Failure to do this means you are working unethically and could lead you to being accused of academic misconduct which could lead to a mark of zero. It is your responsibility to complete or address these outstanding conditions and provide evidence of how you have done this to your supervisor who will then sign the form below. This provides the evidence of how you have addressed the conditions set by the committee and must be included in your final dissertation along with your signed ethics form.

The Committee reminds you at this time that you must not change or stray from this research concept, ensure you adhere to the HESS ethics guidelines and have a completed and signed ethics form and a signed copy of this letter submitted in your final dissertation. Failure to do so will result in a final mark of zero as stipulated in the HESS ethics guidelines.

Committee comments

* Q12. - Bit more depth in protocol needed. I assume MedX will be carried out pre- and post- SAFT90. Is a warm up required? How is this carried out? What cool down is required and will this be supervised?; * Q13. - PARQ and consent forms to be kept in a locked cabinet within CC039; * Q34. - I would say yes to this if data is being collected within the laboratory; * PIS - Spelling of 'experienced'; Is the purpose of this study to examine if fatigue of the musculature will alter the way the participant's sprint? This may be true for Craig's PhD but not for this particular study; I am not sure what he means by saying the participants 'DO NOT have to answer the questions to the medical questionnaire'. They do if they want to take part!; Does he need to include the contact details for his DoS?; * Risk Assessment - All hazards associated with the MedX. I suggest adding some additional hazards related to the SAT90 (e.g. slips, trips and falls);

Scott Burnet
Chair
The Health, Exercise and Sport Science Ethics Committee.

6.2. ETHICAL APPROVAL FOR STUDY 1 (PART 2)

Research Project Amendments

This form should be completed if either of the following points apply:

1. Your ethics application was approved but with conditions, or,
2. You are making a 'minor' amendment(s) to your approved study. A minor amendment is defined as any change that is regarded as having the same or fewer ethical implications than that previously approved in your original application (e.g. changing from a maximal test to a submaximal test, or replacing one questionnaire for another).

If either of the above points apply then you should complete the table below and submit it as follows:

- **Staff:** Email to the Chair of the HESSEC (Scott Burnet)
- **Undergraduate or postgraduate students:** submit to the Research Project Amendment link on the Health, Exercise and Sports Science Ethics Applicants (HESSEA) SOL page.

Name	Craig Perrin
Student Number (if appropriate)	
Full title of study	A study to measure the magnitude of lumbar extensor fatigue experienced in competitive footballers after simulating a football match.
Area(s) of research (physiology, etc.)	Physiology

Detail clearly and concisely how you have: - Addressed the conditions attached to your application or, - The changes you wish to undertake to your study. In both situations you should provide the original condition or approach and how it has been addressed / changed. A clear rationale for the change(s) will also need to be provided. Where appropriate suitable academic references should be used to support the rationale for change.	The originally approved ethics application detailed a study design that included a 90-minute (plus 15 min rest) protocol to simulate the physiological demands of football (SAFT90) with a pre and post measure of isolated lumbar extensor torque in amateur male football players. I wish to amend this ethics document to conduct an identical study that replaces the measure of isolated lumbar extensor torque to one that measures trunk flexion endurance as well as hand-grip strength pre and post the SAFT90 protocol. Specifically, the trunk flexion endurance test will follow McGill, Childs and Liebenson (1999) recommendations. Participants will be seated on a massage bed that is inclined to approximately 60°, and participants hips and knees will be flexed to approximately 90°. The test will commence by asking participants to lift their upper body away from the bed and support their own trunk. Participants will be asked to hold this position for as long as possible and the test will cease once participants can no longer support their own trunk (i.e. their trunk is supported by the bed). For each participant, trials will be filmed using a mobile device to confirm the start and end of the test and its duration. Hand grip will be measured on the dominant hand 3 times using a Handgrip dynamometer (5001 Grip-A, Takei, Japan). References: McGill, S., and A. Childs, and C. Liebenson, 1999. Endurance Times for Low Back Stabilization Exercises. *Arch Phys Med Rahbil*, 80(8), 941–944

HESSEA: Application Approved

Scott Burnet
To Craig Perrin

Reply | Reply All | Forward | ...

Mon 07/12/2020 16:15

Dear Craig,
"A study to measure the magnitude of lumbar extensor fatigue experienced in competitive footballers after simulating a football match"
Date: Monday 7th December 2020
Reference: perrc2020
HESS Ethics Committee Decision – **Approved**
Thank you for submitting your application to the Health, Exercise and Sports Science Ethics Committee (HESSEC). Your ethics submission has been considered by the Committee and the Committee has found there to be no major outstanding ethical issues. Therefore, your study has been approved by the committee.
Regards
Scott Burnet
Chair The Health, Exercise and Sport Science Ethics Committee

Scott Burnet [BSc (Hons), MSc, PGCLTinHE]
Senior Lecturer in Research Methods and Exercise Physiology
Faculty of Sport, Health, and Social Sciences
T: 023 8201 3692

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Please click on the link below to book a tutorial:
https://learn.solent.ac.uk/course/view.php?id=18547

6.3. ETHICAL APPROVAL FOR STUDY 2, 3 AND 4

Health, Exercise and Sports Science Ethics Committee

Outline of proposed research to be submitted for ethical review

PLEASE NOTE: Students will need to discuss this form with their project tutor for each project undertaken. Before completion the applicant is advised to consult the Health Exercise and Sport Science Ethical Policy available on the HESSEC SOL page. In addition, the applicant should also read and understand the British Association of Sport and Exercise Sciences (BASES) Code of Conduct and if the project falls within the auspices of psychology, then you are advised to ensure you have read and understood the Ethical Principles for Conducting Research with Human Participants published by the British Psychological Society.

This form must be completed by Staff, Postgraduates and Undergraduates before potential participants are approached to take part in any research. Please complete all questions.

IMPORTANT: By ticking the following box you confirm that your supervisor has checked your application and is happy for it to be submitted to the Health, Exercise and Sports Science Ethics Committee (HESSEC). If you submit the application and the box is not ticked then your application will be automatically declined. ☒

Student Name:

Craig Perrin

Project Title:

The effects of soccer specific lumbar extensor fatigue on sprint performance and kinematics (Studies 2 and 3).

Area to be studies:

- Biomechanics ☒
- Performance analysis ☐
- Physiology ☒
- Psychology ☐
- Sociology ☐
- Strength and Conditioning ☐
- Nutrition ☐

Please refer to the list of approved procedures in **appendix D of the HESS Ethics document** on the HESSEC SOL page and detail which apply to your study:

Study 2 (lumbar fatigue protocol): Blood pressure will be measured prior to testing (2o) and maximal strength testing of the lumbar muscles will be conducted (3i).

Study 3 (sprint kinematics with lumbar fatigue): Blood pressure will be measured prior to testing (2o). Anthropometric measurements will be taken (2i) followed by the attachment of joint markers using hypoallergenic tape (5b). Following this, the lumbar muscles will be fatigued using resistance exercise (3i) and subjects will be recorded during a 10 m sprint for 3D motion analysis (5a).

Study 4 (knee flexor fatigue after an ILEX fatiguing protocol): Blood pressure will be measured prior to testing (2o). Lumbar muscles will be fatigued, and maximal strength testing of the knee flexors will be conducted (3i).

Proposed duration of study (i.e., from the start of data collection to completing the written report:

01/06/18 – 1/11/18 (approximate)

Contact Number: **07791201502**

Level of study: Postgraduate (MSc / MPhil / PhD)

Supervisor: **James Steele**

Other investigators: **Brian Wink and Dave Smith**

Question 1:

- Does your study involve the use of human participants?
    - Yes
- Minors (under 18 years of age) or vulnerable adults (e.g. adults with specific learning needs)
    - No
- Overt observation techniques, such as notational analysis?
    - No
- Questionnaires or semi-structured interviewing?
    - No
- The discussion of sensitive topics (e.g. drug abuse)?
    - No
- Covert observation or deceptive procedures (i.e. participants are unaware of the purpose of your research)?
    - No

- Sub-maximal exercise testing (i.e. less than 85% heart rate) this may also involve testing flexibility testing, massage techniques?

    - Yes
- Physiological testing that is maximal in nature and greater than that experienced in everyday life? This might include activities such as one repetition maximum testing, time-trialling or maximal exercise testing ($VO_{2\,max}$ test)

    - Yes
- High risk psychological or physiological distress or harm to participants that exceeds normal life. This might include activities such as 1) paramedic or medicine attendance during a resting ECG and throughout experiential testing; 2) clinical trials; 3) research on abnormal or clinical psychology; 4) in addition, participants are children under 5, pregnant women or vulnerable adults:

    - No

Question 2: Concisely describe the research problem / issue under investigation:

Hart et al., (2009) found fatiguing the paraspinal muscles increased trunk flexion during steady state jogging. However, the causation of these findings cannot be solely attributed to paraspinal fatigue as the hip extensors likely contribute to trunk extension when the pelvis can rotate freely (Fisher, Bruce-Low and Smith 2013). Similarly though, fatiguing the lumbar extensors Increased hip flexion whilst standing (Madigan, Davidson and Nussbaum 2006), a task that reduces the variables influencing forward lean in comparison to running. Trunk flexion appears to be a consistent symptom of lumbar extensor fatiguing protocols though none isolates this muscle group. In addition, It is expected that alterations at the spine and hip complex, such as increased trunk flexion, would consequently alter kinematics in the lower limbs. Higashihara et al., (2015) noted increased forward lean during sprinting is accompanied by greater anterior pelvic rotation and flexion at the knee. These changes would have implications for injury risk and potentially impair sprint performance. A common theme throughout aforementioned research is the use of fatiguing tasks that lack external validity to sport. Indeed, whilst it is likely lumbar extensor fatigue induces trunk flexion, it is not known whether the same magnitude of fatigue occurs under sport specific circumstances.
From the available literature, lumbar extensors fatigue may be partly responsible for the kinematic changes whilst fatigued, but current fatiguing protocol fail to isolate the lumbar extensor musculature and use an arbitrary magnitude of fatigue.

Question 3: What is the rationale that underpins the study (i.e. why is the research worth conducting)? Please include theory (i.e. academic literature) in support of your study:

The lumbar extensor musculature is typically of great interest in low back pain research, but its potential to impair
performance is overlooked. The cross-sectional area of the erector spinae and quadratus lumborum are
significantly correlated to sprint performance from 0 - 20m and combined they explain 50% of the variation in velocity over 20m (R2= 0.504; Kubo et al., 2011), yet the reason for such findings are unclear. Considering sprinting is a crucial task in many sports, any possible impairments or alterations to sprint performance should be investigated. Such investigations have the potential to

minimise injury risks and develop athletic performance. This may be particularly important in sports such as soccer, where the injury risk and performance demands are high.

Question 4: Concisely state the aim of the study (i.e. what is the aim of the study?):

The study aims to design an isolated lumbar extensor fatiguing protocol that replicates the lumbar extensor fatigue experienced from soccer (identified in Study 1 – already has ethical approval). Secondly, this protocol will be used to identify differences in acceleration kinematics and performance that can be attributed to soccer related lumbar extensor fatigue.

Question 5: Which methodological approach are you looking to adopt?

- Quantitative

Question 6: Which research design are you looking to adopt?

- Experimental

Question 7: in the box below, please provide details of the questionnaires, protocols, techniques, and procedures to be used during your data collection (e.g. CSAI-2 inventory, Wingate test, capillary blood sampling, and semi-structured interviewing). This should include:

- Warm-up and cool-down procedures
- Testing procedures (e.g. massage techniques, 1 RM protocols, questionnaires to be used, semi-structured interview themes / questions).
- Reliability and validity data of the testing procedures.
- Training procedures to be used (e.g. intensities, durations, and frequency of training).

Study 2:
The MedX Lumbar extension measures lumbar strength isometrically across the individual's full range of motion (normally from 0° to 72° at 12° intervals) and will be executed according to the manufacturer's guidelines. This will begin with dynamic warm up completing 6-8 submaximal repetitions, followed by 3 isometric contractions using 50% effort at full flexion and extension, and neutral (middle range of motion). This will be followed by maximal isometric strength testing over the full ROM. Participants will be allocated 3s to gradually increase force to maximum at each angle. Once maximal strength has been obtained, dynamic repetitions will be performed using either high load and low repetitions, or low load and high repetitions, until the target decrease in force has been achieved. The specific parameters of the protocol will be identified once Study 1 has been completed. After completing the fatiguing protocol, the participant will complete light static stretching of the back supervised by the researcher to ease any discomfort. A second visit may be necessary to repeat this process if the protocol parameters need further refinement.

Study 3:
As above, the MedX lumbar extension device will be used to induce lumbar extensor fatigue based on the protocol obtained in the previous study. Currently, it is expected this will use either high loads and low repetitions, or low loads and high repetitions. Secondly, sprint kinematics will be assessed before and after the lumbar fatiguing protocol. The sprint trial will take place within the biomechanics laboratory over 10m using a standing start. In the event a participant fails to

decelerate, a crash mat will be positioned at the end of the sprint prior to testing. In addition, trainers must be worn and all trip hazards (e.g., tables and chairs) will be removed from the testing area. Prior to any testing, 2 sub-maximal sprints (50 % effort) and 1 maximal sprint will be completed over the testing space for familiarisation and warm-up. Vicon motion capture will be used to record sprint kinematics over a portion of the 10m sprint, and passive joint markers will be attached using hypoallergenic tape according to the full body plug-in Gait model. Additional markers may be added to capture lumbar spine kinematics. After completing the final sprint trial, the researcher will oversee light stretching of the low back and additional stretches will be provided at the participant's request to reduce any muscular discomfort.

Study 4:

Using University students in sports related degrees, knee flexor torque will be assessed after completing the ILEX fatiguing protocol identified from Study 2. Knee flexor torque will be assessed isometrically using the MedX Knee flexion device (MedX, Ocala, FL, USA) pre and post ILEX fatigue. Participants will be sat upright with a knee angle similar to that used during ILEX. A warm-up consisting of 8 dynamic contractions at a self-selected low load under the researchers guidance, followed by 2 sub maximal isometric contractions at 50% effort. For both pre and post-tests, two repetitions will be completed though a 3rd repetition will be conducted if the second exceeds the first. This study will allow us to determine whether additional muscles are fatigued by completing the ILEX repetitions.

Study 4:

Using University students in sports related degrees, knee flexor torque will be assessed after completing the ILEX fatiguing protocol identified from Study 2. Knee flexor torque will be assessed isometrically using the MedX Knee flexion device (MedX, Ocala, FL, USA) pre and post ILEX fatigue. Participants will be sat upright with a knee angle similar to that used during ILEX. A warm up consisting of 8 dynamic contractions at a self-selected low load under the researchers guidance, followed by 2 sub maximal isometric contractions at 50% effort. For both pre and post-tests, two repetitions will be completed though a 3rd repetition will be conducted if the second exceeds the first. This study will allow us to determine whether additional muscles are fatigued by completing the ILEX repetitions.

Question 8: How do you intend to analyse your data (e.g. content analysis, one-way ANOVA)?

The torque loss induced by isolated lumbar extension (ILEX) fatiguing protocol will be compared to the torque loss from the simulation of football using equivalence testing. ILEX induced fatigue will be considered equivalent to soccer related fatigue if the 95% CI of the effect size is within a lower and upper bound effect size. These upper and lower bounds represent effect sizes represent the range of results deemed practically equivalent and will be selected based on the variation of fatigue obtained during soccer simulation in Study 1. If the 95% CI for effect size is within this bound, the fatigue is considered statistically equivalent.

Study 4:

A paired samples t-test or Wilcoxon-signed rank test will be used to compare for pre and post differences in knee flexor torque with alpha set at 0.05.

Question 9: Briefly describe the sample demographic and the sample method. This should include:

- Sampling procedure (e.g. convenience, purposive, theoretical)
- Characteristics of the participants you wish to use (e.g. gender, age)
- Participant inclusion and exclusion criteria (e.g. inclusion - must play competitive sport; exclusion: lower limb injury in the last 6 mth)

Participants will be purposively sampled to be male, adults (over 18yrs), and competitive footballers. Participants must not have any malignancy or underlying disease, disc herniation, osteoporosis, neurologic or sciatic nerve root compression, previous vertebral fractures, major structural abnormality of the spine, problems passing fluid or solids and inflammatory arthritis. Furthermore, participants must not currently be undergoing rehabilitation or possess any lower limb injury.

For study 4, the same criteria will be adhered to with the exception of being university students on sports related degrees rather than competitive footballers.

Question10: Methods of recruiting participants (e.g. poster, social media, team briefing etc.)

Individuals who meet requirements of the study will be given the opportunity to take part via email, poster advertisement, or word of mouth, without external pressure for participants to take part. All participants will receive a full description of the study via an information sheet so participants have clear expectations of their role. In addition, contact details will be given to provide clarification by email or phone if necessary. This communication will be informative only and not used to encourage participation. Local football clubs are expected to be the main source of recruitment.

Question 11: Where will the study take place (e.g. university, physiology laboratory, a named school, a named hospital etc.)?

Solent University's Physiology and Biomechanics laboratories (Southampton).

Question 12: Give an estimate of the amount of time you will require of each participant in the study / project. Please break this down into potential sub-headings like briefing, testing / training, debrief etc.

- Briefing:
- Testing (include the frequency and duration):
- Training (include the frequency and duration):
- Debrief:

Study 2:

Visit 1:

- Medical questionnaire (PARQ) and briefing (10 minutes)
- MedX lumbar extensor device set up and dynamic warm up using submaximal loads (5 minutes)
- Lumbar extensor baseline strength testing across full ROM (2 minutes)
- Fatiguing protocol (approximately 2 minutes)
- Lumbar extensor strength testing post fatigue (2 minutes)

- Low back stretches supervised by researcher and debrief (5 minutes)
- Buffer time for unexpected delays (5 minutes)
- Total time: 31 minutes

Visit 2 (if necessary):
- As above
- Total time: 31 minutes

Study 3:
- Medical questionnaire (PARQ) and briefing (10 minutes)
- Anthropometric measurements and joint marker application (20 minutes)
- 3 warm up sprints (2 minutes)
- Sprint trial at rest (2 minutes)
- Removal of 6 joint markers for lumbar extensor exercise and travel from biomechanics lab to physiology lab (3 minutes)
- MedX lumbar extensor device set up and dynamic warm up using submaximal loads (5 minutes)
- Fatiguing protocol and travel to biomechanics lab (approximately 2 minutes)
- Re-application of joint markers (5 minutes)
- Post-fatigue sprint trial (3 minutes)
- Removal of joint markers (5 minutes)
- Post testing stretches and de-brief (5 minutes)
- Buffer time for unexpected delays (15 minutes)
- Total time: 1 hr 20 minutes

Study 4:

- Medical questionnaire (PARQ) and briefing (10 minutes)
- Knee flexor warm up (8 dynamic repetitions using a self-determined low load followed by 3 submaximal isometrics (50% effort) (4 minutes)
- Rest (1 minute)
- Knee flexor baseline strength testing (2 minutes)
- Fatiguing protocol (approximately 2 minutes)
- Knee flexor strength testing post lumbar fatigue (2 minutes)
- 5-minute buffer for unexpected delays
- Total time: 26 Minutes

Question 13: How do you plan to handle the requirement of participant confidentiality (e.g. password protected laptop or file)?

All data will be kept on a password-protected computer and will only be available to the relevant researchers. In addition, individual participants will be assigned a code to ensure anonymity. Paper documents, including PARQ's and consent forms, will be secured in a locked cabinet within CC039. After 2 months, from the completion of the research, any data that could be used to identify the participants will be discarded.

Question 14: Does your study have the potential for "upsetting" participants (e.g. affective manipulation) and/or for identifying distressed or disturbed individuals? If 'Yes', you must make

"a priori" arrangements to mitigate such effects (e.g. debriefing). Please specify the nature of such arrangements, if required, on a separate piece of paper.

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 15: Do you intend to pay participants for their participation?

- No

Question 16: Will you be using any form of deception (i.e. the participants are unaware of the study aims)?

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 17: Will the study require the cooperation of a gatekeeper (e.g. coach or teacher) for initial access to the groups or individuals to be recruited (e.g. school pupils, residents of nursing homes)? This is a letter **FROM THE GATEKEEPER AND NOT A LETTER TO THE GATEKEEPER.** A letter to the gate-keeper is **NOT** acceptable. Please scan and attach the letter with this application. The letter should be on headed paper from the gatekeeper OR in the form of an email from an official email address.

- No

Question 18: Will it be necessary for participants to take part in the study without their knowledge and consent at the time (e.g. covert observation of people in public places)?

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 19: Will the study involve the discussion of sensitive topics?

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 20: Are drugs, placebos or other substances (e.g. food substances, vitamins etc.) to be administered to the study participants or will the study involve invasive, intrusive or potentially harmful procedures?

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 21: Will blood or tissue samples be obtained from participants?

- No

- If yes, please provide details (e.g. type of blood sampling, approximate number of samples, frequency of sampling): Click here to enter text.

Question 22: Is pain or mild discomfort likely to result from the study? If so, how will the effects be moderated?

- Yes ☒
  - Comment: Click here to enter text.
- No ☐

Question 23: Is there any risk to participants (physical and, or psychological) greater than that normally experienced in normal life? If so, please comment.

- Yes ☒
  - Comment: Click here to enter text.
- No ☐

Question 24: during your data collection will supervision or assistance be required (e.g. for experiments in the physiology laboratory)? If so, please comment.

- Yes ☐
  - Comment: Click here to enter text.
- No ☒

Question 25: Unless there are very good reasons, informed consent (possibly assent) will have to be obtained? ***Please copy and paste your consent form (possibly assent form if you are working in minors) into the box below*:**

Informed Consent

Name of experiment: The effects of soccer specific lumbar extensor fatigue on sprint kinematics and performance

1. I can confirm that the full details of the experiments/investigations have been explained to me. I am clear about what will be involved and I am aware of the purpose, the potential benefits, and the potential risks. I can also confirm that I have had the opportunity to ask questions that I have about the experiment/investigation procedure.

2. I recognise that I have the right to withdraw my involvement at any time during the testing procedure.

3. Any data collected and stored on a computer will remain anonymous, however I understand that complete anonymity cannot be safeguarded due to the public nature of laboratory sessions.

4. I have completed a health questionnaire and agree to take part in this study.

Name of Participant Participants Signature Date
…..... …….....…….

Declaration by the Academic Investigator/Project Officer

I can confirm that I have provided detailed information about the procedure that the above participant has consented to.

Name of Staff **Staff Signature** **Date**

…….. **……..…….**

Question 26: Will a medical questionnaire need to be administered (e.g. Physical Activity Questionnaire – PAR-Q)?

- Yes

Question 27: Will a pre-study questionnaire need to be administered (e.g. International Physical Activity Questionnaire)?

- No

Question 28: Does your project involve using children as your participant population? If 'yes', for children under the age of 18, their own consent (where possible) and parental / guardian consent is required this must to written consent). Please enter your DBS number.

- Yes ☐
  - DBS Number: Click here to enter text.
- No ☒

Question 29: Will your project require access to special populations (e.g. physically impaired / mentally impaired) as your participant group?

- No

Question 30: Is parental / guardian consent required for your project? If 'yes' please enter the form in the box below.

- Yes ☐
  - If yes, enter parental / guardian consent form below: Click here to enter text.
- No ☒

Question 31: If undertaking questionnaire based research are you aware that you are required (i.e., this is not optional!) to have an ID card (your campus card will suffice) on show at all times and if collecting your data off campus you will be required to work in pairs (to maintain a safe environment) as well as informing one of your peers of your leaving time, location of destination and expected return time. By ticking yes you are agreeing to undertake this task.

- No

Question 32: Are frequent and repeated checks on your participants required during the data collection (e.g. heart rate, rate of perceived exertion etc.)?

- No

Question 33: Is a first aider required to be available during data collection?

- Yes

Question 34: Are you appropriately competent or qualified to undertake the testing **AND** training? Is a **letter of competence** (e.g. from tutor or coach) or evidence of competence (e.g. REP's or coaching certificate) required? This is required if you are undertaking specific procedures such as **laboratory testing** (e.g. Wingate tests, incremental exercising testing, blood sampling), **massage / therapy-based procedures** (e.g. FMS, MET) or **gym-based testing** (e.g. 1 RM testing).

- Yes ☒
  - If yes, please scan the letter or email and attach it to your submission
- No ☐

Question 35: Is a participant information sheet (PIS) required? If so, please enter the PIS here.

- Yes ☒
- No ☐

Please copy and paste your participant information sheet (PIS) into the box below:

Attached below.

Adobe Acrobat Document

Question 36: Please complete the risk assessment form below. This may include

- Physical and psychological risks associated with the testing and training (e.g. over-exertion from testing / training or trauma associated from questioning – career ending injury).
- Physical risks associated with the location (i.e. is the environment safe to collect data)
- Your safety if collecting data off-site (e.g. travelling to participants away from the University)

Hazard		Project		Supervisor		Date	
Look out for hazards which you could reasonably expect to result in significant harm under the conditions in your study. Provide a full description.							
E.g. Injury	**Is risk adequately controlled?**	**Risk rating**			**Who might be harmed?**	**Is further action necessary to control the risk?**	**Residual level of risk**		
Look out for hazards which you could reasonably expect to result in significant harm under the conditions in your study. Provide a full description.	What precautions have already been undertaken?	Probability	Severity	Rating	Tick the groups of people who are especially at risk from significant hazards which you have identified	List the actions you will take where it is reasonably practicable to more	Probability	Severity	Rating
E.g. Injury Soft tissue injury from sprinting or knee flexion strength testing	*Subjects will be required to thoroughly warm up prior to all test procedures*	*2*	*2*	*4*	*Staff* *Students* *Others*	*Perform warm up following the ACSM (2007) guidelines*	*2*	*1*	*2*
Soft tissue injury from sprinting or knee flexion strength testing	Subjects will warm up, gradually increasing the intensity, prior to testing.	2	2	4	Staff Students Others	Participants cannot be undergoing rehabilitation or currently suffer from a lower limb injury	1	2	2

MedX Machinery malfunction										
MedX Machinery malfunction Spine injury	The researcher is trained and competent at using the equipment. Protocols will be aligned with the manufacturer guidelines.	1	1	1	Staff Students Others	 ✓ 				
Spine injury Low back muscle injury	The exclusion criteria prevents injury to the spine.	1	4	4	Staff Students Others	 ✓ 				
Low back muscle injury Slips, Trips, Falls, and Collisions	Lumbar muscles will be warmed up appropriately.	2	3	6	Staff Students Others	 ✓ 	Subjects will perform the lumbar extension exercise with correct technique through familiarisation trials prior to initial testing.	1	3	3
Slips, Trips, Falls, and Collisions Allergies	Appropriate footwear should be warn at all times and the area will be checked for wet and slippery areas. Crash mats will be placed at the end of the sprint and any tables, chairs, or	2	2	4	Staff Students Others	 √ 				

	loose wires/equipment will be removed from the testing area.									
Allergies	Hypoallergenic tape will be used for marker placement.	1	3	3	Staff					

- **PROBABILITY X SEVERITY = RISK RATING** - **1-4** = The risk is low and adequately controlled
- **5-8** = Review controls, take additional action if required- **9, 12, 16** = **DO NOT** UNDERTAKE ANY TESTING/DATA COLLECTION! Urgent action is required to reduce the risk!

Question 37: Please complete the following insurance form.

The purpose of this form is to decide whether the University requires additional insurance cover for a clinical/research trial. The form should be fully completed, and returned to the Programme Administrator.

This questionnaire should be completed for every research project that will involve human participation.

Name of Sponsor (usually Southampton Solent University): **Solent University (Southampton)**

Title of Research: The effects of soccer specific lumbar extensor fatigue on sprint kinematics and performance.

Number of participants: **Approximately 38**

Is your research going to be based upon the following?

- Questionnaire or interviewing:
  - No
- Venepuncture:
  - No
- Measures of physiological processes:
  - Yes
- Collections of body secretions by non-invasive methods:
  - No
- The administration of mouth of foods or nutrients or variation of diet other than the administration of drugs or other food supplements or psychological activity (this is outside the research definition:
  - No

Is the research to be held in the UK? If it's not, then please provide details.

- Yes ☒
  - Please provide details: Click here to enter text.
- No ☐

Who will be involved in conducting the research? Student (Craig Perrin)

If medical practitioners are involved will they be covered by the Medical Defence Union (MDU) or any other organisations?

- Yes ☐
- No ☒

Does the research involve use drugs or surgery?

- Yes ☐

- No ☒

Are any of the research participants (after enquiry) known to pregnant?

- Yes ☐
- No ☒

Are any of the research participants (after enquiry)?

- Yes ☐
- No ☒

Is the purpose of the research to?

- Investigating or participating in the methods of contraception:
  - Yes ☐
  - No ☒
- Assisting with, or altering the process of contraception:
  - Yes ☐
  - No ☒

Does the research involve genetic engineering?

- Yes ☐
- No ☒

Will the research use pharmaceutical product designed or manufactured by the university?

- Yes ☐
- No ☒

Proposed commencement date AND the proposed duration of the research:

- Start date: 01/06/2018
- Duration: 5 months (approximately)

Will the sponsor pay for additional insurance costs if required?

- Yes ☐
- No ☒

If other organisations are involved in the research, is SSU the lead organisation for the research project?

- Yes ☐
  - Please provide details: Click here to enter text.
- No ☒

Dear Craig,

HESS Ethics Committee Decision – APPROVED with Conditions

"The effects of soccer specific lumbar extensor fatigue on sprint performance and kinematics (Studies 2 and 3)**"**

Reference: perrc2018

Thank you for your ethics application to the Health, Exercise and Sports Science Ethics Committee (HESSEA). Your ethics submission has been considered by the Committee and no major outstanding ethical issues have been identified. **However**, there are several important conditions set out below which you must meet and are set out below. The Committee expects you to liaise with your Director of Study to discuss these conditions and to ensure they are addressed prior to your data collection. Failure to do this will mean that you are working unethically and could lead to the implementation of academic misconduct procedures. It is your responsibility to complete or address these outstanding conditions and provide evidence to your supervisor on how you have done this.

The Committee reminds you at this time that you must not change or stray from this research concept. Please ensure you adhere to the HESSEA guidelines and include the following in your Project write-up.

- The electronic version submitted to the committee
- This email, signed by your project supervisor confirming you have addressed the outstanding conditions (prior to data collection), acknowledging the HESS Ethics Committee's decision

Therefore, please file this email appropriately and **DO NOT** delete it. Failure to include it in your project may result in a final mark of zero as stipulated in the HESS ethics guidelines
Conditions:

- Rationale could be supported more with evidence.

- Perhaps need to be clearer on the loads/repetitions to be used for MedX lumbar extension - how will these be ascertained?

- How will sprint effort be ascertained?

- Q21 - Include details of blood sampling

- Q22 - How will effects of mild pain be moderated?

- Q23 - Risk to participants?

Regards

Scott Burnet

Chair - The Health, Exercise and Sport Science Ethics Committee.

Student Signature: 

Project Supervisor Signature: 

Project Supervisor Name: James Steele

6.4. EXAMPLE PARTICIPANT INFORMATION SHEET

PURPOSE

The aim of this study is to identify the extent muscles of the low back are fatigued from completing a football match. Secondly, to understand whether the same amount of fatigue alters the way footballers sprint, and ultimately how fast they sprint. Sprinting is a vital component within football and we wish to investigate the possibility that low back fatigue may impair sprint performance and even whether an increase in inury risk may occur.

INVITATION

You have been invited to take part as you are male, an outfield footballer with no current lower limb injuries. As this study has a specific focus on fatigue during football, it is important the tests are also conducted on outfield football players. Unfortunately, for this reason goalkeepers are excluded from this study as they have a different physical role. Before making a desicion to take part, it is important you are fully informed of your potential role. The following text should take no more than 10 minutes of your time - if you have any questions or if something is not clear, feel free to ask and we will be happy to answer them for you.

THE STUDY

THE EFFECTS OF LOW BACK MUSCLE FATIGUE ON SPRINT PERFORMANCE

WHAT TO EXPECT

STEP 1: SIMULATE FOOTBALL FATIGUE

Step 1 will require you to attend the university laboratories on 2 occasions. The first visit will be approximately 1 hour and consist of familiarising yourself with the equipment used to measure back strength. This test requires you to sit within a device where you will push backwards against a pad using your back with maximal effort for 3 seconds. This test is repeated at a variety of angles usually from 0° (leaning fowards) to 72° (leaning backwards) at 12° intervals.

Also, you will perform the first 15 minutes of an indoor protocol used to simulate the physical demands of a football match without a ball and opponents. The protocol consists of repeateadly covering 15 metres at a variety of speeds (e.g., walking, running, sprinting) alongside side-stepping manouveres.

The second visit will begin with a measure of back strength, followed by the full football simulation protocol (90 minutes), and a final back strength measurement to examine any decreases in force, representing fatigue. This visit will be approximately 2 hours.

STEP 2: REPLICATING FATIGUE

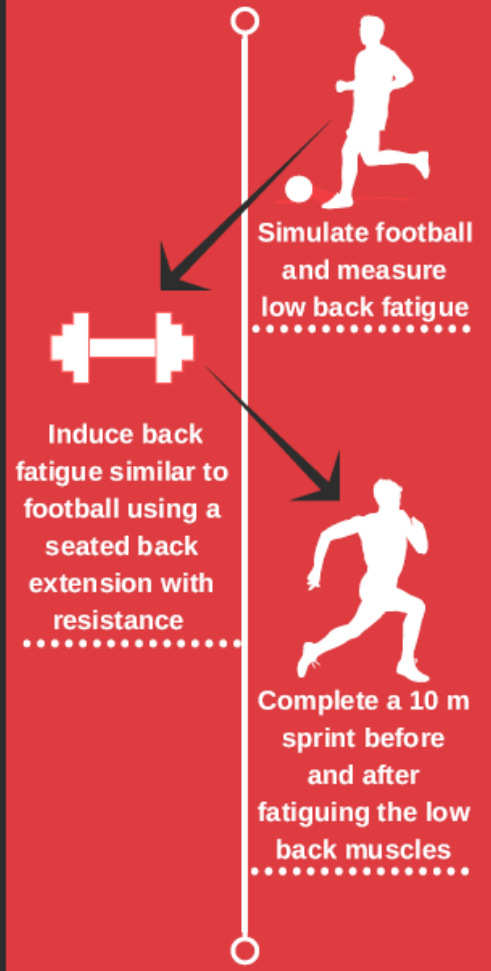
To understand the role of back muscle fatigue in sprinting, it is crucial that only the back muscles are fatigued when assessing sprint technique. For this reason, simulating a football match would be inappropriate as multiple muscles would also be fatigued in addition the low back muscles.

Instead, the seated back extension, used in the first step, will be used to induce low back fatigue to a similar extent to the fatigue experienced from football simulation.

The precise details of the fatiguing task (e.g., heavy or light weights, low or high repetitions) will be dependent on the amount of fatigue experienced during simulated football - identified in the first step. Broadly, you should expect to perform light weights for many repetitions or heavy weights for fewer repetitions. Repetitions will be performed from leaning forwards to leaning backwards in the seated position.

The exact details will be provided as soon as we know them. This is likely to be after sufficient participants have completed Step 1. Step 2 should consist of 1 visit to the laboratory, though a second visit may be necessary to refine the fatiguing protocol to better replicate football fatigue. The session will take no more than 30 minutes.

THE PROCESS



STEP 3: EFFECTS ON SPRINTING

Using the procedure identified in Step 2 to induce fatigue in the low back muscles, we can now observe how fatigue in these muscles alters sprinting.

First, you will complete a 10 m sprint at rest. This allows for a comparison to be made against sprinting with fatigue. Next, you will complete the fatiguing procedure identified in Step 2 to induce football-related back muscle fatigue. Immediately after, a second sprint will be completed to compare changes in sprint performance.

Sprint performance will be assessed using several joint markers attached to the skin using double-sided tape. These are easily removed and unlikely to cause any discomfort. In some circumstances, light shaving of the skin (14 mm diameter) may be necessary to ensure adhesion between the marker and the skin. The joint markers will allow a computer-generated 3D model of your sprint to be created, allowing precise measurements of any changes that occur from low back fatigue. Step 3 requires a single visit approximately 1 hr and 20 minutes in duration.

Prior to all tests, a medical questionnaire and blood pressure reading will be conducted to ensure your safety. Trainers and shorts are required for all sessions.

AFTERWARDS

Once the research stops the data will be analysed appropriately. You will not be required for any further testing for this study however, if you have any questions please feel free to contact us at any time. If you wish to know the results of this study please let us know and again we will be pleased to inform you.

EXPENSES

There are no expenses to take part in the study however, it will require you to travel to Southampton Solent University on approximately 4 occasions.

BENEFITS

By taking part in this study, we will be a step closer to understanding the role of the low back muscles in athletic performance.
In doing so, you will gain an insight into the laboratories that are frequently used by professional athletes and sports teams, experience some of the tests they undergo using equipment woth £1000's. In addition, we are happy to give you any personal results from the tests you complete.

FURTHER INFORMATION

POSSIBLE SIDE EFFECTS

It is possible, although unlikely, you may feel some muscular discomfort within the low back in the following days of each study however this is likely to subside after a few days and should not interfere with your day to day life. In the first study, you will experience some fatigue in the lower limbs but this will be similar to what is experienced after a regular football match.

DO I HAVE TO TAKE PART?

It is up to you to decide whether to take part. If you do decide to take part, you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are free to withdraw at any time and without giving a reason. If you do decide to withdraw we would like to stress this will not influence your treatment and you shall still receive a full debrief and ask any questions if you wish.

WHO HAS REVIEWED THE STUDY?

All research from the University in the Health, Exercise and Sport Science Programme is looked at by the HESS ethics committee, to protect your interests. This study has been reviewed and passed.

PRIVACY

We will follow ethical and legal practice and all information about you will be handled in confidence. The data gained from this study will be kept on a secured, password protected laptop and data will only be shared with the relevant researchers. All names will be replaced with codes and so participants will remain anonymous. Data that could be used to identify you will be kept for 2 months after the study to allow for appropriate analyses but afterwards this data will be deleted permanently.

CONTACT DETAILS

If you have any questions regarding the study whatsoever, please feel free to contact me at Craig.Perrin@solent.ac.uk.

If you have any health concerns we recommend you discuss these with an appropriate healthcare professional before making a decision.

Thank you for your time.

REPORTING CONCERNS

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed by a researcher instantly. If you remain unhappy and wish to complain formally, you can do this via Scott Burnet, Chair of the HESS Ethics Committee, Faculty of Business, Sport and Enterprise at Southampton Solent University (Scott.Burnet@solent.ac.uk or 02382013692).

WANT TO TAKE PART?

If you wish to be a part of this research please contact the lead investigator, Craig Perrin, to arrange a date for testing. You can contact Craig via email at: Craig.Perrin@solent.ac.uk.

NOTE: Once arrangements have been made, failing to attend without any prior notice may prevent you from taking part in future related research.

6.5. EXAMPLE CONSENT FORM

Name of experiment:

1. I can confirm that the full details of the experiments/investigations have been explained to me. I am clear about what will be involved and I am aware of the purpose, the potential benefits, and the potential risks. I can also confirm that I have had the opportunity to ask questions that I have about the experiment/investigation procedure.

2. I recognise that I have the right to withdraw my involvement at any time during the testing procedure.

3. Any data collected and stored on a computer will remain anonymous, however I understand that complete anonymity cannot be safe guarded
due to the public nature of laboratory sessions.

4. I have completed a health questionnaire and agree to take part in this study.

Name of Participant	Signature	Date
.....	.....	.....

Declaration by the Academic Investigator

I can confirm that I have provided detailed information about the procedure which the above participant has consented to.

Name of Staff	Signature	Date
.....	.....	.....

6.6. PHYSCAL ACTIVITY READINESS FORM

All information provided will remain confidential

Name.....

Date of Birth..... Age..... B.P.....mmHg

How would you describe you current level of fitness? Unfit/moderately fit/trained
Are you currently a smoker? Yes/No
Are you a previous smoker? Yes/No

Do you drink alcoholic drinks? Yes/No
If yes do you have: the occasional drink? Yes/No
a drink every day? Yes/No
more than one drink a day? Yes/No

Do you suffer, or have you suffered from
Asthma (within 2 years)? Yes/No
Diabetes? Yes/No
Bronchitis? Yes/No
Epilepsy? Yes/No
Any form of heart complaint? Yes/No
Dizziness or fainting? Yes/No

Is there any history of heart disease in your family?
Yes/No – If yes give details

.....

Do you currently have any form of muscle or joint injury that may be aggravated by the testing?
Yes/No – If yes give details

.....

Have you had any cause to suspend normal activity in the last two weeks?
Yes/No – If yes give details

.....

Are you currently taking any form of medication?
Yes/No – If yes give details

.....

In the past month, have you had chest pain when you were not doing physical activity?
Yes/No – If yes give details

.....

Do you feel pain in your chest when you do physical activity?
Yes/No – If yes give details

.....

Are you suffering from infectious skin diseases, sores, or blood infections (i.e. Hepatitis B, HIV etc.)?
Yes/No – If yes give details

.....

Have you had hyper/hypothermia, heat exhaustion, or any other heat or cold disorder?
Yes/No – If yes give details

.....

Have you had anaphylactic shock symptoms to needles, probes or other medical-type equipment?
Yes/No – If yes give details

.....

Have you had chronic or acute symptoms of gastrointestinal bacterial infections (e.g. Dysentery, Salmonella)?
Yes/No – If yes give details

.....

Do you have a history of infectious diseases (e.g. HIV, Hepatitis B); and if appropriate to the experimental design, have a known history of rectal bleeding, anal fissures, haemorrhoids, or any other condition of the rectum?
Yes/No – If yes give details

.....

Do you have any allergies to plasters, micropore tape, skin electrodes or latex gloves?
Yes/No – If yes give details

.....

Finally, do you know of any other reason that may prevent you from participating in physical activity?
Yes/No – If yes give details

.....

Please supply a name and contact number of a person we can contact in the event of an emergency

Name.....

Telephone number.....

Participant's Signature..... Date.....

Staff Signature..... Date.....

Repeat tests only

I can confirm that my answers to this PARQ have not changed since the previous test and that I am happy to take part in the repeat tests.

Participant's Signature..... Date.....